THE CANADIAN MEDICAL ASSOCIATION

LE JOURNAL DE

L'ASSOCIATION MÉDICALE CANADIENNE

AUGUST 13, 1960 • VOL. 83, NO. 7

CONGENITAL CARDIAC MALFORMATION IN THE NEWBORN PERIOD FREQUENCY IN A CHILDREN'S HOSPITAL*

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AN APPROXIMATION of the incidence of congenital cardiac malformations in the population has been reached by different workers in a variety of ways.[1-4] A prospective study conducted by Richards and colleagues[2] found a figure of about seven congenital cardiac malformations per 1000 live births. Because of admitted sources of error inherent in any study of such large numbers of infants over a short period of one year, this figure was regarded by the authors as "almost certainly low". It has been suggested that between 15% and 30% of infants born with cardiac malformation die in the first month of life.[2-4] Regardless of the accuracy of this estimate, the fact is established that a substantial number of infants with congenital heart disease die in the neonatal period.

While the problem of determining an accurate incidence of unspecified congenital cardiac malformation is great, the difficulty of arriving at precise information on the frequency of the different defects in affected infants is greater. For varying reasons the frequency of the different malformations has differed greatly in several reports. The present study does not pretend to solve this dilemma. In expressing the recent experience of congenital heart disease in the first 28 days of life from a children's hospital with a large referral population, we approach the problem in a manner different from most previous communications.

CASE MATERIAL AND METHODS

Records were examined of all infants under the age of 29 days referred to the cardiac service of the Hospital for Sick Children, Toronto, between 1953 and 1957, together with all autopsies within the same age group where congenital cardiac malformation was found in the same five-year period. A total of 264 such infants was divided into two main groups.

(a) Infants dying with cardiac malformations established by post-mortem examinations:
- Operative deaths 24
- Non-surgical deaths 106

(b) Infants assessed in, but surviving, the newborn period
- Pseudo-cardiopathy[5], i.e. patients referred with possible heart disease in whom no cardiac malformation was found 53
- True congenital cardiac malformation 81

RESULTS

The Fatal Group

The 24 patients who died at or shortly after operation during the first month of life were separated for the reason that while in almost every instance death might have been expected without surgical treatment, a number could have survived 28 days. The chief proportion had transposition of the great vessels (seven patients), preductal coarctation of the aorta (six patients) and pulmonary atresia with normal aortic root (four patients).

The frequency of the major anatomical defect at autopsy in 106 patients who died in the neonatal period is shown in Table I. Malformations above

TABLE I.—THE FREQUENCY OF DIFFERENT CARDIAC MALFORMATIONS AT AUTOPSY IN 106 NEWBORN INFANTS BETWEEN 1953 AND 1957

	Per cent
Aortic atresia or stenosis	23
Coarctation of the aorta	13
Transposition of the great vessels	10
Pulmonary atresia or stenosis	8
Ventricular septal defect	7
Atrioventricularis communis	5
Tetralogy of Fallot	5
Complicated dextrocardia or levocardia	5
Mitral atresia or stenosis	4
Complete anomalous pulmonary venous drainage	4
Single ventricle	3
Persistent truncus arteriosus	3
Endocardial fibroelastosis	2
Ebstein's disease	2
Atrial septal defect	2
Patent ductus arteriosus	2
Tricuspid atresia	2
Total	100

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the black bar constitute together about 75% of the total number. Over one-third of the deaths were associated directly or indirectly with aortic lesions and more than half of the deaths were in infants with one of four malformations. Approximately two-thirds of the deaths were in the first week of life. Eight per cent were dead on arrival at the Admitting Department while 12% were not referred to the cardiac service, chiefly being examples of preductal coarctation of the aorta. In 4% of the total, heart disease was unsuspected before autopsy, and again these were chiefly aortic lesions or a ventricular septal defect. A single case of transposition of the great vessels was discovered at autopsy.

About one-quarter of the babies who died were premature, and one-fifth died from causes other than their cardiac malformation. The etiological agent in these cases was infection, prematurity or other gross malformations. Forty-four per cent of the group developed congestive heart failure before death. Fourteen per cent, i.e. 20% of those available, were investigated by accessory methods of study, such as cardiac catheterization or angiocardiography.

The Surviving Group

Of 149 infants seen by us with signs of heart disease in the first month of life who survived that period, 81 had true cardiac malformations and 53 pseudocardiopathy. A further 15 were seen and diagnosed as having heart disease, but since their fate after discharge was not known, they were excluded from the study. Of this latter number, ventricular septal defect, transposition of the great vessels and atrioventricularis communis were present in about equal numbers.

TABLE II.—DIAGNOSIS IN 53 NEWBORN INFANTS WITH PSEUDO-CARDIOPATHY, I.E. PATIENTS PRESENTING WITH SIGNS SUGGESTING HEART DISEASE IN WHOM NO CARDIAC MALFORMATION WAS FOUND

	Percentage
Respiratory distress syndrome	16
Innocent murmurs	15
Disappearing ventricular septal defect	12
Disorders of heart rate	6
Positional cardiac abnormalities	2
Extrathoracic AV fistula	1
Systemic hypertension, undetermined	1
Total	53

The pseudocardiopathies constitute a group the size and content of which vary in different institutions, but in our instance, in this five-year period, 53 patients had the diagnoses given in Table II. During the time of the study, cases of respiratory distress were largely confined to the mature babies with cyanosis or babies in whom frank congestive heart failure was present or suspected. The innocent murmurs were chiefly of hæmic origin in infants with blood incompatibility disorders. Those infants with so-called disappearing ventricular septal defect presented with a characteristic murmur but no other abnormalities. The murmur disappeared between six weeks and two years later. Evidence now suggests that this syndrome is due to a minute defect in the muscular septum, but these patients are not included as ventricular septal defects for the purposes of this study because complete proof of this assumption is still lacking. Supraventricular tachycardia or congenital heart block accounted for the cases with disordered heart rate, and dextrocardia for those positional anomalies of the heart without heart disease.

TABLE III.—THE FREQUENCY OF DIFFERENT CARDIAC MALFORMATIONS IN 81 INFANTS EXAMINED DURING AND SURVIVING THE NEWBORN PERIOD

	Percentage
Ventricular septal defect	20
Tetralogy of Fallot	16
Transposition of the great vessels	16
Pulmonary stenosis or atresia	13
Coarctation of the aorta	11
Complicated dextro- or levocardia	5
Single ventricle	4
Atrioventricularis communis	4
Ebstein's disease	2
Patent ductus arteriosus	2
Tricuspid atresia	2
Persistent truncus arteriosus	2
Mitral stenosis	2
Aortic stenosis	Less than 2
Total	100

Eighty-one infants remain who were seen in the newborn period and found to have true malformation of the heart (Table III). The malformations above the black bar constitute together about 75% of the total number. The malformation which headed the list in the fatal group is now the least common. Ventricular septal defect and tetralogy of Fallot assume a position of greater importance, while transposition of great vessels, pulmonary stenosis and coarctation of the aorta hold their previous positions. It will be noted that over half of the infants in this group were suffering from one of four malformations, namely, ventricular septal defect, tetralogy of Fallot, transposition of the great vessels or pulmonary stenosis. Sixteen per cent of the infants in this group were premature, and 25% developed congestive failure during the period of follow-up. In the same period, of the 81 infants, 31 died, 60% in the first six months and 90% by the end of the first year. Three-quarters of the babies were investigated by cardiac catheterization or angiocardiography, and the majority (73%) were so studied during the first month of life. Surgical treatment was undertaken in 20 of the 81 patients. Not all were treated in the first month and only half were operated upon under six months of age. There were five deaths, including one patient who died 21 months after operation. The 15 survivors included seven cases of coarctation of the aorta,

two of pulmonary stenosis, two of ventricular septal defect, two of tricuspid atresia, one of tetralogy of Fallot and one of patent ductus arteriosus.

DISCUSSION

Observations previously reported on the type of congenital cardiac malformation in newborn infants have shown considerable differences in frequency. The difficulty of obtaining accurate information from the various methods of study has been well appreciated by the several authors. Theoretically the ideal method is prospective analysis from a nursery population, but in one institution the chances of obtaining a large enough sample in the lifetime of one group of observers is remote. Smaller numbers followed for shorter periods end in figures of dubious value. Retrospective observations of frequency based on infants undergoing autopsy in a maternity institution over a very long period of time suffer the double handicap of documentation by examiners of differing experience or interest in congenital cardiac malformation as well as excluding infants dying after being sent home or transferred to a specialized unit elsewhere because of cardiorespiratory distress. At the other extreme, the information from children's hospitals tends to overemphasize the fatal malformations which are referred early. It does not necessarily reflect the true incidence of different malformations unless the institution receives all infants in the area in cardio-respiratory distress and unless the actual mortality figures correspond with the predicted figure from the number of live births in that area. The particular interests of the cardiac unit have a considerable bearing on the number of early referrals of infants with signs of heart disease but in no distress. Calculations based on live births and neonatal deaths in the city of Toronto during the period of the present study suggest that barely 50% of the estimated number of newborn infants dying with congenital cardiac defects were examined in the Hospital for Sick Children. Further, only one infant in every six born with heart defect was actually assessed in the first month of life in the same period of time.

The chief interest in congenital malformation in the newborn period lies in those patients in whom the malformation in the natural course of events leads to death during this period. Of this particular segment, experiences from Boston,[6] Scandinavia[7] and the present series show that of those malformations responsible for two-thirds of all fatalities in the newborn period, transposition of the great vessels and ventricular septal defect occupy a prominent place, the list being completed by coarctation of the aorta, aortic atresia, pulmonary stenosis and tetralogy of Fallot. The variation occurs in the order of precedence. The only really uniform proportion in these three studies lies in the frequency of coarctation of the aorta, which occurs in between 11 and 13%. In contrast, no cases of aortic atresia were noted in the Boston series, and only 9% were noted in the Scandinavian study. In the present report it was the leading fatal malformation, accounting for almost one-quarter of the total deaths. Over the five-year period of the Toronto study there was an average of five to six per year, and since the study was closed, although the numbers with aortic atresia are a little higher each year, the proportion of the total deaths from cardiac malformation in the neonatal period has remained about the same. For example, in 1958, aortic atresia was found in 10 of 30 autopsies in newborn infants with cardiac malformation while in 1959 the corresponding numbers were 8 and 38. Although one-third of the patients at autopsy are from outside metropolitan Toronto, the revised frequency figures for the city area alone do not show any important change in the percentage of the leading malformations.

Of the conditions in infants who have signs of cardiac malformation but who survive the newborn period, ventricular septal defect appears to be the most frequent. The proportion of cases of this lesion would be even higher (32% instead of 20%) if those patients with the characteristic disappearing murmur had been included rather than placed in the group of pseudocardiopathies. There is mounting evidence that spontaneous closure of small defects of the muscular ventricular septum is responsible for the clinical picture of disappearing murmurs of this type.[8] Tetralogy of Fallot, as well, is found in an increasing proportion of the surviving patients, while pulmonary stenosis and coarctation of the aorta remain at about the same relative frequency.

Despite some variations in the frequency figures of congenital heart malformation at all ages in large hospital experiences,[9-14] a composite suggests that ventricular septal defect and patent ductus arteriosus are the most common defects, followed by atrial septal defect, tetralogy of Fallot, pulmonary stenosis and coarctation of the aorta. These six malformations account for about 75% of affected individuals.

The surviving group of newborn patients reported here represents, in a sense, a transition between the very different incidence of fatal malformations in the neonatal period and those in older patients. The survivors include a high proportion (60%) with ventricular septal defect, tetralogy of Fallot, pulmonary stenosis and coarctation of the aorta, but differ principally in having a high incidence of transposition of the great vessels and a low incidence of patent ductus arteriosus and atrial septal defect. It is interesting that in newborn infants the ductus arteriosus, while an important temporary component of the respiratory distress syndrome and a secondary malformation in a wide variety of other cardiac malformations, is not usually a serious problem as an isolated lesion. The same is true of isolated atrial septal defects.

The ultimate aim of a study of this sort is to attempt to show which major malformations are contributing to the high mortality of congenital cardiac disease in very early life as well as to indicate the differing presentation of less severe malformations in this period. It would be unrealistic to suggest that the results of this study are valid for all areas and institutions or even to claim that they are more than an approximation of the true incidence. The figures do, however, suggest that a relatively limited number of regularly recurring malformations accounts for the main problems in this age group in hospital practice. They form a basis upon which attempts by family physicians, cardiologists and surgical teams can be made to improve the present discouraging situation.

SUMMARY

Of 211 infants with congenital heart malformation who were examined during the first month of life in a children's hospital over a five-year period, 130 died during the first month and 81 survived beyond 28 days.

An additional group of 53 patients with signs initially suggesting heart disease were found on further study to have no cardiac malformation. The majority of fatalities were accounted for by aortic atresia, coarctation of the aorta, transposition of the great vessels and pulmonary atresia or stenosis with normal aortic root, in that order.

The commonest lesions in the surviving patients were ventricular septal defect, tetralogy of Fallot and transposition of the great vessels.

It is suggested that the majority of patients with heart disease presenting in the newborn period are accounted for by a relatively small number of malformations.

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RÉSUMÉ

Pendant cinq ans à l'hôpital des enfants malades de Toronto, on a examiné, au cours de leur premier mois de vie, 211 nourrissons porteurs de malformation congénitale du cœur. De ce nombre, 130 moururent avant d'atteindre la cinquième semaine et 81 survécurent au-delà de 28 jours. Après une observation plus poussée, un autre groupe de 53 enfants chez qui on avait soupçonné la présence de cardiopathie s'en avérèrent indemnes. La plupart des mortalités relevèrent des causes suivantes, en ordre d'importance: l'atrésie aortique, la coarctation de l'aorte, la transposition des gros vaisseaux avec atrésie pulmonaire ou la sténose pulmonaire avec une souche aortique normale. Chez les survivants les lésions les plus fréquentes furent les défauts de la cloison ventriculaire, la tétralogie de Fallot et la transposition—des gros vaisseaux. Il ressort de cette étude que la majorité des affections cardiaques du nouveau-né ne tiendrait qu'à un nombre relativement restreint de malformations.

DES PROTEINURIES

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I. L'ALBUMINURIE LORDOTIQUE PHYSIOLOGIQUE

NOUS AVONS observé 130 sujets sains, tous de sexe masculin, âgés de 7 à 22 ans, non porteurs d'albuminurie permanente. L'épreuve d'orthostatisme simple a permis de découvrir, parmi ces 130 sujets, six cas d'albuminurie orthostatique.

Chez les 124 autres garçons, nous avons pratiqué, après miction et absorption de 150 à 200 c.c. d'eau, l'épreuve d'hyperlordose orthostatique d'une demi-heure. A la fin de l'épreuve, le sujet urine. L'albuminurie, lorsqu'elle est découverte, est évaluée en milligrammes par minute. Seules sont naturellement retenues les albuminuries pathologiques, c'est-à-dire supérieures à 0.12 mg. par minute.

Sur les 124 sujets, la lordose orthostatique a fait apparaître l'albuminurie dans 58 cas (soit 46.7%) à des taux variant de 0.20 à 40 grammes par litre, la plupart des résultats se situant entre 2 et 10 g./l. (soit 23 cas), des taux supérieurs à 10 g. étant notés dans 10 cas. Evaluée en mg./min., cette albuminurie lordotique se situe entre 0.20 mg./min. et 1.0 mg./min. dans 22 cas, entre 1 et 5 mg./min. dans 27 cas et est supérieure à 5 mg./min. dans 9 cas (5 mg./min. représentent théoriquement une perte de 7.20 g./24 h.). La protéinurie est donc importante dans plus de la moitié des cas. L'analyse électrophorétique montre que cette protéinurie est mixte: 70 à 80% d'albumine, une faible proportion d'a_1 et β globulines, des traces d'a_2 et γ globulines.

L'étude de la répartition des cas d'albuminurie lordotique en fonction de l'âge montre une augmentation progressive du pourcentage des sujets albu-

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minuriques en lordose en fonction du nombre d'années, passant de 20% chez les sujets de moins de 10 ans, à 92% dans le groupe de 14-16 ans. Chez l'adulte jeune, par contre, on retrouve un pourcentage faible: 14%.

L'âge, facteur important en apparence, n'est pas le facteur essentiel; celui-ci est la puberté: si les enfants, tous impubères avant 10 ans, les impubères de plus de 10 ans, ont une albuminurie lordotique, en fait, entre 14 et 16 ans, 83% des enfants qui sont en plein développement pubertaire présentent cette même albuminurie, contre 39% chez les sujets encore impubères à cet âge. L'ensemble de la statistique montre, d'ailleurs, que les garçons en plein développement pubertaire, quel que soit leur âge, ont facilement une protéinurie lordotique: 85% contre 33% chez les sujets n'ayant pas encore apparemment commencé leur puberté et 14% chez les jeunes adultes ayant, en apparence, terminé la leur.

Si l'on considère, enfin, le débit-minute des sujets en lordose, on constate que les garçons porteurs d'une albuminurie lordotique ont une nette diminution de leur diurèse par rapport au groupe des sujets non albuminuriques: débit en moyenne inférieur à 1 c.c./min. dans le premier cas, supérieur à 2 c.c./min. dans le second, mais cette réduction de la diurèse ne suffit pas à expliquer l'albuminurie, puisque, évaluée en mg./min., la protéinurie est de très loin supérieure à l'albuminurie physiologique. Albuminurie et oligurie doivent donc dépendre de la même cause, sans doute circulatoire. En conclusion, chez 85% des garçons, en période pubertaire, on peut déclencher une protéinurie pathologique par hyperlordose.

II. Les Albuminuries Orthostatiques

Seules sont orthostatiques les albuminuries que provoque la position debout, qui disparaissent en position couchée et qu'aucune autre circonstance ne peut faire apparaître.

Sur 130 adolescents sains, nous avons découvert six albuminuries orthostatiques, nous l'avons vu plus haut. D'après notre statistique, l'albuminurie orthostatique constitue un quinzième des albuminuries isolées. Nous avons examiné 100 sujets atteints d'albuminurie orthostatique; leur âge moyen est de 15 ans et tous ont moins de 25 ans (sauf deux de 26 et 46 ans).

L'étude d'une albuminurie soupçonnée orthostatique nécessite des dosages sur les urines émises en diverses positions, avant et après repos, avec vérification entre chaque épreuve de l'absence totale d'albuminurie en clinostatisme. De cette étude il découle que l'albuminurie orthostatique est moindre après un effort physique même violent et prolongé qu'après une station debout prolongée immobile (par exemple: 25 g. % après 30 minutes d'immobilité, 5 g. % après deux heures de marches, 0 après 30 minutes de repos couché). L'albuminurie orthostatique peut varier de 0.50 à 25 g. %.

Cliniquement ces malades sont 14 fois sur 15 des adolescents des deux sexes en plein développement pubertaire, flasques, hypotoniques, longilignes. La lordose provoquée lorsqu'elle n'est pas déjà évidente accentue l'albuminurie en position debout. Les épreuves rénales sont bonnes et la numération des éléments figurés urinaires est normale en position couchée. Le test à la glace est négatif dans 96% des cas. L'urographie révèle assez souvent (11 fois sur 15) une ptose rénale uni ou bilatérale.

La correction de la ptose par le port d'une ceinture correcte fait disparaître l'albuminurie en position debout (1 fois sur 3) mais il va de soi que l'on voit chez les adolescents hypotoniques des ptoses rénales sans albuminurie orthostatique. Cette albuminurie orthostatique isolée est d'évolution bénigne et guérit avec les années. Lorsque cette albuminurie orthostatique s'accompagne de signes de néphrite *a minima* ou constitue la séquelle d'une néphrite aiguë, elle guérit dans 80% des cas. Le traitement de l'albuminurie orthostatique consiste, lorsqu'elles sont importantes, en la réduction médicale de la ptose rénale et la correction de la lordose, mais par prudence il est sage de supprimer chirurgicalement les foyers infectieux rhinopharyngés lorsqu'ils existent.

L'albuminurie orthostatique s'accompagne d'oligurie, mais il n'y a pas de parallélisme entre l'oligurie et le taux de protéinurie; 50% des albuminuries orthostatiques ont une clearance du mannitol basse et 90% de ces patients ont une clearance du P.A.H. normale. L'étude de l'hémodynamique rénale de ces sujets en décubitus, puis en lordose orthostatique nous a montré que:—les clearances en position couchée sont toujours normales;—la lordose orthostatique entraîne une chute immédiate de la filtration glomérulaire (de 20 à 67%) et du flux plasmatique rénal (de 18 à 83%) et une augmentation de la fraction de filtration. Plus l'orthostatisme est prolongé, plus la dépression des clearances est accentuée. Après le retour en clinostatisme, les chiffres de filtration glomérulaire et du flux plasmatique rénal reviennent à la normale en 18 à 30 minutes.

La diurèse diminue instantanément en lordose orthostatique (de 11.1 à 1.50 c.c./min. en moyenne). L'oligurie persiste après le retour en décubitus dorsal. L'élimination urinaire des électrolytes s'effondre également en position debout. L'apparition de l'albuminurie coïncide exactement avec la diminution de la filtration glomérulaire et du flux plasmatique rénal, les débits urinaires maxima d'albumine correspondent assez étroitement avec les amenuisements les plus marqués du flux plasmatique (3-4 mg./min.). La disparition de l'albuminurie correspond au moment où la filtration glomérulaire et le flux plasmatique rénal rejoignent leur valeur de contrôle. Dans un cas où le flux plasmatique est resté abaissé en position couchée après l'épreuve d'orthostatisme, sans dépression du filtrat glomérulaire, l'albuminurie a persisté jusqu'à

la réascension du flux plasmatique aux chiffres antérieurs normaux.

En conclusion, l'albuminurie orthostatique propre à l'adolescent au moment de la puberté est bénigne et nous semble indépendante des variations de la diurèse, relativement indépendante des variations de la filtration glomérulaire, habituellement liée aux variations du flux plasmatique rénal.

III. Les Albuminuries Permanentes Isolées

L'albuminurie chronique permanente apparemment isolée a été étudiée avec notre maître le Professeur Pasteur Vallery-Radot chez 200 patients. Elle exige d'abord d'être confirmée dans son existence: urates, mucine, phosphates, pseudo-albumine acéto-soluble, albumines dégradées, albumose doivent être éliminés. L'albuminurie peut être le fait d'une pyurie, mais celle-ci doit alors être massive. L'albuminurie peut être le fait d'une hématurie, mais pour qu'il en soit ainsi il faut que l'hématurie atteigne 50,000,000 de globules rouges par centimètre cube d'urine.

La découverte d'une albuminurie permanente ne doit pas conduire à admettre une corrélation absolue et certaine de cause à effet entre la maladie qui a fait découvrir l'albuminurie et cette maladie elle-même—si l'albuminurie n'a pas été recherchée au préalable à plusieurs reprises et dans un délai rapproché. La découverte d'une albuminurie doit par contre faire rechercher une infection rhinopharyngée ou cutanée récente et parfois encore en évolution.

On sait l'intérêt que revêt, pour connaître la date probable d'apparition d'une albuminurie, la notion d'une recherche négative d'albuminurie antérieurement, à l'occasion d'une vaccination, d'une grossesse, du service militaire, d'une visite d'embauche. Les malades ont souvent perdu le souvenir de cette analyse et l'interrogatoire la leur remémore. Si, après cet examen, la vaccination a été pratiquée, la mise au régime sans sel non demandée, le service militaire accompli, l'embauche acceptée, cela signifie que le malade était indemne d'albuminurie.

On sait aussi la valeur qu'il faut accorder à la présence d'abondants flocons mousseux à la surface des urines; ils correspondent à une albuminurie importante. La date de leur constatation permet de préciser approximativement le début ou la date d'aggravation de la maladie. Enfin une bouffissure du visage, dont l'apparition a pu être notée par l'entourage, a également de la valeur. Outre les signes de déficience rénale, d'infection urinaire, d'hypertension, l'interrogatoire recherche la notion d'une albuminurie chez les proches.

L'examen du malade révèle exceptionnellement une maladie polykystique, une maladie du col vésical ou de la prostate. La cause réelle est pratiquement toujours ignorée, mais l'on découvre dans 33% des cas une hypertrophie amygdalienne avec adénopathie, dans 20% des végétations adénoïdes hypertrophiques et infectées, dans 3% une infection cutanée, et dans 1.5% une sinusite chronique, une otite chronique, une infection dentaire.

L'accentuation de l'albuminurie est appréciée selon la position, selon l'alimentation, lors d'épisodes infectieux. L'albuminurie doit être considérée en fonction du volume urinaire et du nycthémère, donc en milligrammes par minute. La numération des éléments figurés de l'urine, les fonctions rénales, le bilan humoral, l'état cardio-vasculaire, l'urographie constituent des temps nécessaires. L'urographie révèle une fois sur 10 des anomalies rénales.

Le traitement de l'albuminurie permanente n'est pas encore connu. Nous n'avons guéri qu'une seule fois une albuminurie par amygdalectomie. Nous n'avons vu que deux fois sur 200 régresser spontanément et définitivement une albuminurie permanente.

Le pronostic de l'albuminurie permanente chronique isolée est difficile à établir. Cependant, séquelle d'une néphrite aiguë, elle peut être cicatricielle et disparaître à la longue ou persister indéfiniment sans aggravation; plus souvent, apparue insidieusement sans que l'on puisse en découvrir la cause, elle évolue vers l'insuffisance rénale avec ou sans hypertension.

D'après nos 200 cas, nous pensons pouvoir conclure que: 1° l'albuminurie paraît ne pas évoluer dans 47% des cas; 2° la complication la plus fréquente, la plus précoce est l'hypertension artérielle que l'on constate dans 30% des cas; 3° l'insuffisance rénale biologique avec hypertension secondaire ou concomitante survient dans 21% des cas; 4° l'insuffisance rénale sans hypertension artérielle au cours de l'évolution est exceptionnelle (2%); 5° trois cas suivis plus de 30 ans ont tous trois évolué vers l'hypertension artérielle et l'insuffisance rénale.

En conclusion l'albuminurie permanente isolée est donc un symptôme de grave signification pronostique dans un peu plus de la moitié des cas. Nous n'en connaissons le plus souvent pas la cause, jamais le traitement.

IV. Les Albuminuries de la Grossesse

D'après l'étude de 300 femmes ayant présenté de l'albuminurie gravidique il nous semble qu'il n'existe pas une albuminurie gravidique, maladie autonome, mais plusieurs variétés d'albuminurie gravidique:

L'albuminurie physiologique de la grossesse ne s'observe qu'au cours du travail, ne dépasse jamais 0.50 g. % et a la même valeur que l'albuminurie d'effort du sujet sain.

L'albuminurie proprement gravidique survient en fin de grossesse et est presque toujours associée à des œdèmes et à une hypertension artérielle, cette triade symptomatique caractérisant la toxémie gravidique pure. Cette albuminurie, qui a surtout une signification pronostique, disparaît quelques jours ou au maximum quelques mois après l'accouche-

ment et ne récidive habituellement pas lors des grossesses ultérieures.

L'albuminurie gravidique récidivante s'associe quelquefois à une hypertension artérielle modérée, récidivante ou permanente.

Les albuminuries antérieures à la grossesse sont constatées dès le début de la gestation. Elles peuvent revêtir trois types cliniques: (a) les albuminuries isolées en rapport avec une albuminurie primitive ou avec une albuminurie cicatricielle secondaire à une néphrite; (b) les albuminuries avec troubles du fonctionnement rénal traduisant une néphrite chronique; (c) les albuminuries avec œdème diffus.

L'albuminurie résiduelle d'une néphrite aiguë est de beaucoup la plus sévère (85% des cas d'aggravation). L'albuminurie de la grossesse secondaire à une albuminurie transitoire de l'enfance est de beaucoup la plus bénigne (aggravation ou accidents pathologiques dans 14% des cas).

En conclusion, quelle que soit l'étiologie, ces albuminuries antérieures à la grossesse ont deux caractéristiques essentielles: dans 70% des cas, l'albuminurie augmente pendant la grossesse et une hypertension, des œdèmes et une insuffisance rénale s'y associent; l'hypertension artérielle associée à une albuminurie a une action très fâcheuse sur la vie du fœtus dans 40% des cas.

RÉSUMÉ

La protéinurie lordotique s'observe chez 85% des garçons en période de développement pubertaire. La protéinurie orthostatique est curable et semble liée à une chute de la filtration glomérulaire et du flux plasmatique rénal en position debout.

La protéinurie permanente isolée n'est pas actuellement curable. Elle évolue dans 21% des cas vers l'insuffisance rénale biologique lentement progressive puis vers l'hypertension artérielle. Dans 30% des cas elle se complique d'hypertension artérielle avant que n'apparaisse l'insuffisance rénale biologique. Dans 2% des cas elle est à l'origine d'une insuffisance rénale sévère sans hypertension artérielle associée.

La protéinurie gravidique groupe les protéinuries purement gravidiques et les protéinuries antérieures à la grossesse. Dans l'un et l'autre cas la gravité pronostique semble dépendre de l'existence d'une hypertension artérielle associée mettant en péril la vie du fœtus dans 40% des cas.

(Travail du Centre de Recherches sur l'H.T.A.—Chaire de Pathologie Médicale, Faculté de Médecine de Paris—Hôpital Beaujon, Clichy, Seine—Professeur P. Milliez.)

INTRA-ARTERIAL OXYGEN INSUFFLATION IN THE TREATMENT OF PERIPHERAL VASCULAR INSUFFICIENCY: PRELIMINARY REPORT ON 117 CASES*

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THE PRESENTATION of this paper is perhaps premature. However, the clinical benefits of treatment have been so striking that it is felt that a report would be of general interest.

This method of treatment is not new, although I (G.A.C.) must confess complete ignorance of this treatment until January of last year, when it was brought to my attention by a local business man who had been advised to have his leg amputated in 1951 for diabetic gangrene of his foot. He had received treatment in the Möller Clinic in Kassel, Germany, in 1951, and again in 1956. The beneficial results of this treatment interested me enough to take the trip to Germany in February 1959. The results that were obtained at that clinic could not help but impress one.

In February 1959, with the permission of Dr. C. C. Ross, Chief of Service, Surgery, at Westminster Hospital—the Canadian Department of Veterans Affairs Hospital in London, Ontario—we began treating peripheral vascular lesions of the lower extremities, including the most desperate cases.

The treatment used is as follows. The patients are ambulant. Three times a week, oxygen in varying amounts from 20 to 100 c.c. is injected into alternate femoral arteries at the rate of 20 c.c. in five seconds—the amount of oxygen used varies with the patient's tolerance. At the time of intra-arterial injection the patient also receives 40 c.c. subcutaneously in the affected limb.

The injection is made through a 21-gauge, short, bevelled 1½" needle, without the use of local anæsthesia. After the treatment the patient is instructed to apply pressure over the injection site to limit hæmatoma formation, and to remain in the horizontal position for 20 minutes to prevent syncope.

During the injection the patient will describe a feeling of tension in the limb, beginning im-

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mediately at the site of the injection and rapidly travelling to the popliteal region, then more slowly down the leg to the foot. Exactly the same sensation is produced in a person with an above-knee amputation. During this time the leg becomes deadly white, cold and pulseless. This stage lasts from 5 to 15 minutes, then bright red patches appear over the skin and gradually enlarge till the whole limb is bright red, except for small scattered cyanotic patches. The skin temperature may be elevated as much as 12° C. above that previous to injection and the patient will state that he has a pleasant warm feeling in the leg. The opposite leg to the one injected does not go through the first phase but goes through the second phase to a lesser degree. The reaction on the side of injection extends to the costal margin.

After injection some patients complain of headache, an urge to defæcate or urinate, or severe pain in the extremity treated. The volume and rate of injection appear to influence these. However, the above can occur when the minimal amount of oxygen is introduced at a very slow rate. On two occasions patients were treated when suffering from gastroenteritis, and the treatment was followed by extremely severe abdominal colic and vomiting.

Möller's intra-arterial insufflation apparatus is used for injecting the oxygen. With this apparatus the pressure and rate of introduction of the oxygen is easily controlled. There is also a manometer in the system that registers the intra-arterial pressure, thus preventing injection into the femoral vein.

We have treated 117 patients—arteriosclerotic, senile and diabetic, Buerger's syndrome, and postphlebitic syndrome with stasis ulcer. Their ages have varied from 35 to 87 years.

CASE 1.—Mr. V., aged 41, gave a history of coronary occlusion in November 1956, following which he suffered from angina, both on exercise and at rest, and required 20-25 nitroglycerine tablets a day and 2-4 during the night.

The patient developed claudication in the latter part of 1958, and by May of last year it had progressed till he could take only seven steps. He also complained of pain at night in both calves and feet since March of last year.

Treatments began on May 2, 1959. After the third treatment the patient required no nitroglycerine for his angina, and his claudication distance had increased to 200 paces. After 16 treatments he was completely free of pain on exercise and at rest, and he has remained so.

His *femoral arteriogram* showed good filling of the superficial femoral to its bifurcation.

CASE 2.—Mr. H., aged 42, on January 30, 1959, slipped, striking his left ankle with his right foot, which caused a slight abrasion that developed into two infected ulcers with a sinus joining the two.

He was treated by bed rest, antibiotics and foot baths. The infection was controlled, but the ulcers persisted. This patient's feet were cold and cyanotic,

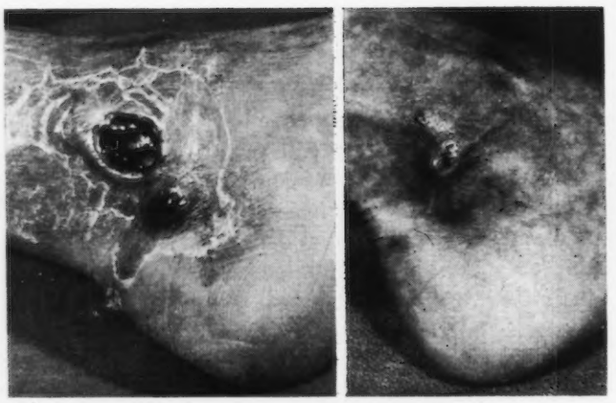


Fig. 1. Fig. 2

Fig. 1.—Case 2—Taken at the beginning of treatment. Shows two ulcers joined by a sinus.

Fig. 2.—After 16 treatments the ulcers healed and have remained so.

and there was no palpable dorsalis pedis pulse on either foot.

On February 19, 1959, intra-arterial oxygen treatment was started and the patient was encouraged to walk around the surgical block. After 16 treatments the ulcer was healed, and his feet were warm and had good colour.

His *femoral arteriogram* showed a normal artery to and including the popliteal.

CASE 3.—Mr. D., aged 68, was admitted to hospital complaining of constant pain in both legs. His claudication distance was nil. His legs were cyanotic, cold and pulseless below the femoral on either side. On the medial side of the right ankle there was a large ulcer of five months' duration. After 34 treatments the patient's claudication distance was over 2000 paces and he was free of night pain. Oscillometric readings before commencement of treatment were nil, both at the knees and ankles. After 30 treatments with the oscillometer set at 100 mm. of mercury there was a suggestion of a pulse at both ankles and a reading of 1 at both popliteals. Temperature readings showed the dorsum of both feet to be 2° C. below our control. The right ankle was the same as the control. The left ankle was 1° higher. The right calf

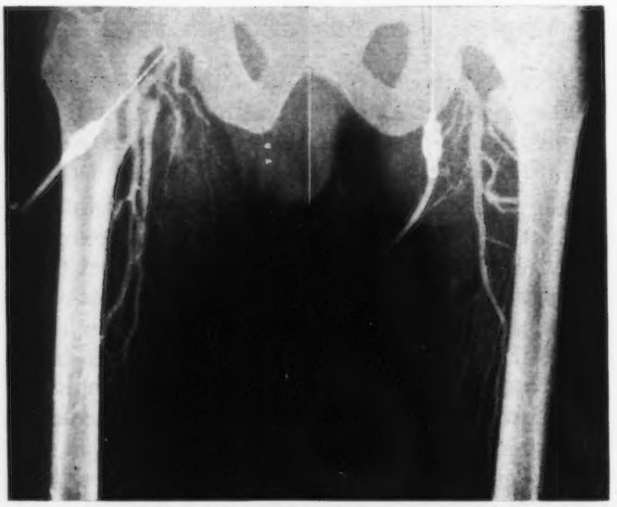


Fig. 3.—Case 3—Arteriograms. Show obstruction of both femoral arteries.

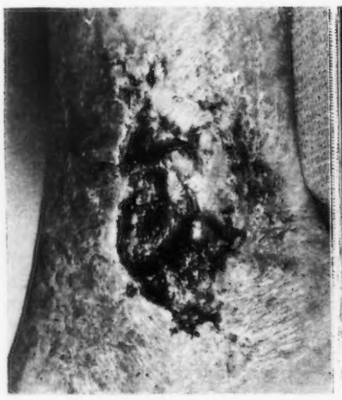


Fig. 4a

Fig. 4b

Fig. 4a.—Case 3—Taken at beginning of treatment. Shows a deep ulcer 2½″ × 1½″.

Fig. 4b.—Taken after 34 treatments. Shows the ulcer healed.

was 1° higher than the control, and the left calf 2° higher than the control. The control patient was a girl laboratory technician, who we assume has normal circulation.

CASE 4.—Mr. S., aged 35, had a guillotine below-knee amputation in 1944. His problem has been recurrent ulceration of his stump on wearing his prosthesis. The patient was encouraged to walk with his prosthesis from the beginning of his treatment. He had a series of 18 treatments.

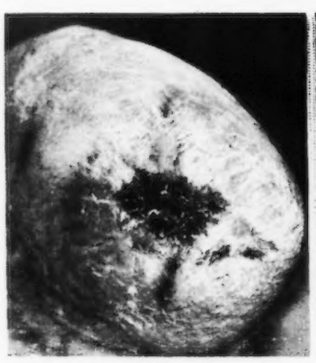


Fig. 5a

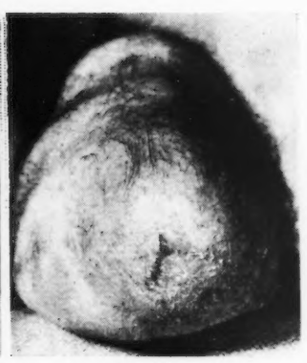


Fig. 5b

Fig. 5a.—Case 4—Taken before treatment. Shows ulceration and scar tissue.

Fig. 5b.—After treatment: The greater part of the scar has been replaced by healthy skin and the ulcer healed. The profuse hair growth is very obvious in this patient. This is the patient's only complaint. (This growth of hair and nail has been noted in almost all cases treated.)

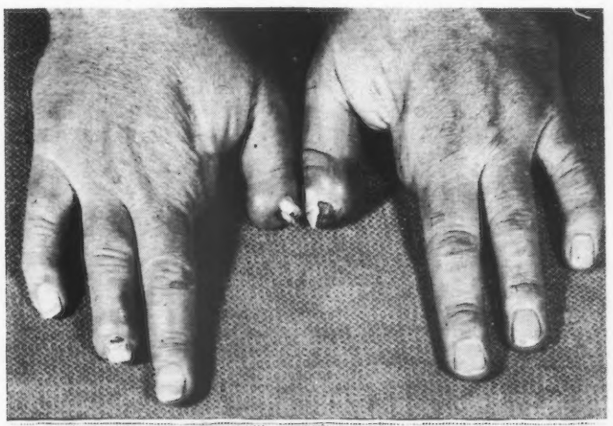
CASE 5.—Mr. R., aged 37, first had symptoms when he was 27. Since then he has had: (a) bilateral lumbar sympathectomies; (b) right mid-thigh amputation; (c) left Syme's amputation; (d) both index fingers amputated.

When he was admitted to hospital he complained of severe pain in his left leg and pain and stiffness of both hands. He had a chronic pulp space infection of both thumbs.

After 24 treatments that included injecting oxygen subcutaneously into both hands, the patient's left leg was warm and painless, as were his hands, and he was able to walk with his crutches.

The results we have obtained from this form of treatment have been very promising—of 117 cases

Fig. 6a



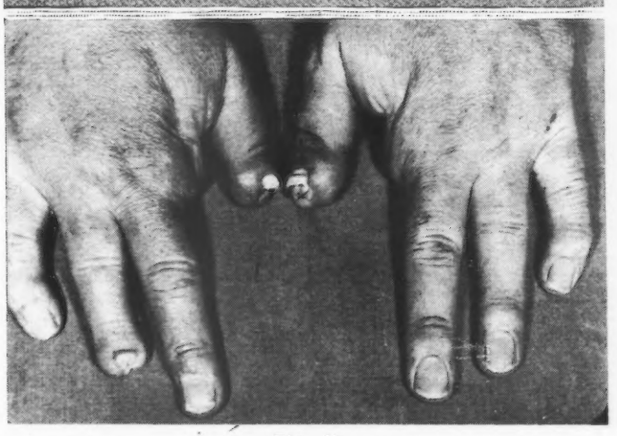


Fig. 6b

Fig. 6a.—Case 5—Hands at the beginning of treatment.

Fig. 6b.—Hands after 24 treatments.

only seven have required amputations. As yet we feel it is too early to classify the results further.

Age, cardiac lesions and other debilitating diseases do not contraindicate treatment.

As yet we have had no complications from repeated puncture of the arterial wall.

We have found that oscillometric readings do increase, and a certain number of patients have a return of pulses.

Skin temperature is elevated and remains so for several months following treatment.

SUMMARY

This preliminary report represents a pilot clinical study of the application of intra-arterial and local oxygen injection treatment for limb ischæmia. No explanation is offered at this time to clarify the altered vascular state producing these favourable results. However, the nature of these results is such as to encourage continued application of this procedure, and studies have begun to elucidate the mechanism responsible for these changes.

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RÉSUMÉ

L'auteur expose sa technique de traitement de l'insuffisance vasculaire périphérique au moyen d'insufflation intra-artérielle d'oxygène. Le malade se présente trois fois par semaine à la clinique où on lui injecte de 20 à 100 cc dans l'artère fémorale à raison de 20 cc dans cinq secondes. On ajoute également, 40 cc d'oxygène par voie sous-cutanée dans le membre atteint d'insuffisance vasculaire. Le malade ressent une tension dans le membre, qui débute à l'endroit même de l'injection, s'étend à la région poplitée, pour enfin atteindre lentement la jambe et le pied. La jambe devient blanche, froide et semble inanimée. La circulation paraît se rétablir 5 à 15 minutes plus tard, alors que des plages rouge vif apparaissent sur la peau, s'étendent et se fusionnent pour englober toute la jambe. Des 117 malades ainsi traités, sept seulement ont dû être amputés. L'auteur n'offre aucune explication pour les résultats bienfaisants de cette thérapie.

FAMILIAL ACIDOSIS OF RENAL TUBULAR ORIGIN*

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RENAL TUBULAR acidosis, a clinical syndrome resulting from impaired renal tubular control of acid-base metabolism, is a disorder whose etiology and pathogenesis are incompletely known. Cases in childhood are believed to result from an inborn error of metabolism. In adults, this syndrome has been associated with pyelonephritis, and bilateral ureteral transplantation into the colon, and observed after administration of sulfonamide drugs and acetazolamide. Other cases have been described, however, in which no causal factor has been demonstrated.[1] The interrelationships between the childhood and adult forms of this disorder have not been clearly elucidated, because of incomplete knowledge of the genetic transmission of the defect. Familial occurrence in the adult has rarely been reported.[2]

It is the purpose of this paper to present observations on the three apparently healthy children (B.W., D.W. and J.W.) of a 33-year-old patient with renal tubular acidosis, whose case history was the subject of a previous report.[3]

MATERIAL AND METHODS

Each of the three children was admitted to the pædiatric ward of the Jewish General Hospital. During the entire hospital stay they were given a diet which provided 200 mg. of calcium daily but was otherwise unrestricted. The control subject, a member of the intern staff, was treated similarly. A 24-hour urine sample was collected as a control after three days on the diet. Ammonium chloride was then administered as the free salt, mixed with jam, in doses of 2 g. every six hours (containing 150 mEq. ammonium) daily for four days, except in the case of J.W. where 1 g. every six hours (75 mEq. ammonium) daily was given. Serial determinations of blood Cl, CO_2, pH and urine Ca^{++}, ammonia and pH were carried out. Twenty-four-hour urine specimens were collected in bottles containing 10 ml. of 1N hydrochloric acid, and individual specimens for pH determination were collected under neutralized mineral oil. Directly after pH measurement, the remaining urine was added to the 24-hour specimens.

Urine pH was measured within 15 minutes of voiding, using the Astrup pH meter. Venous blood for pH determination was drawn anaerobically with a siliconed syringe, without tourniquet, after soaking the arm in water at 40° C. for five minutes and the measurement made under anaerobic conditions with the Astrup pH meter. Plasma CO_2 combining power was measured by the volumetric method of Van Slyke, and urinary ammonia by aeration followed by electrometric titration. Urinary calcium determinations were performed by the Clark-Collip modification of the Kramer-Tisdall method.[4]

RESULTS

Blood pH was normal in all three children and the control subject at the start of the investigation. This was unchanged by ammonium chloride loading in the control subject but was diminished to values of 7.30 in B.W., and 7.28 in D.W. In J.W., who received a smaller dose of ammonium chloride (4 g. daily) because of her age, no change in blood pH occurred.

The serum choride level was elevated at the onset of the study in each of the three children. The values were 108, 109, and 109 mEq./l. There was no appreciable increase in these values with

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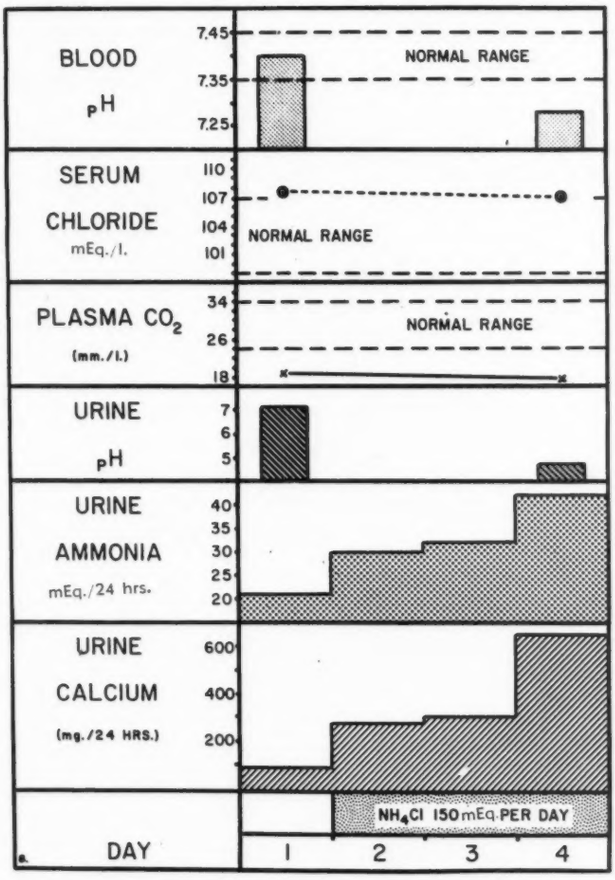

Fig. 1.—Biochemical findings in B.W., showing presence of compensated hyperchloræmic acidosis and alkaline urine. Minimal increase in urinary ammonia with profound hypercalciuria and lowering of blood pH occurred with ammonium chloride loading.

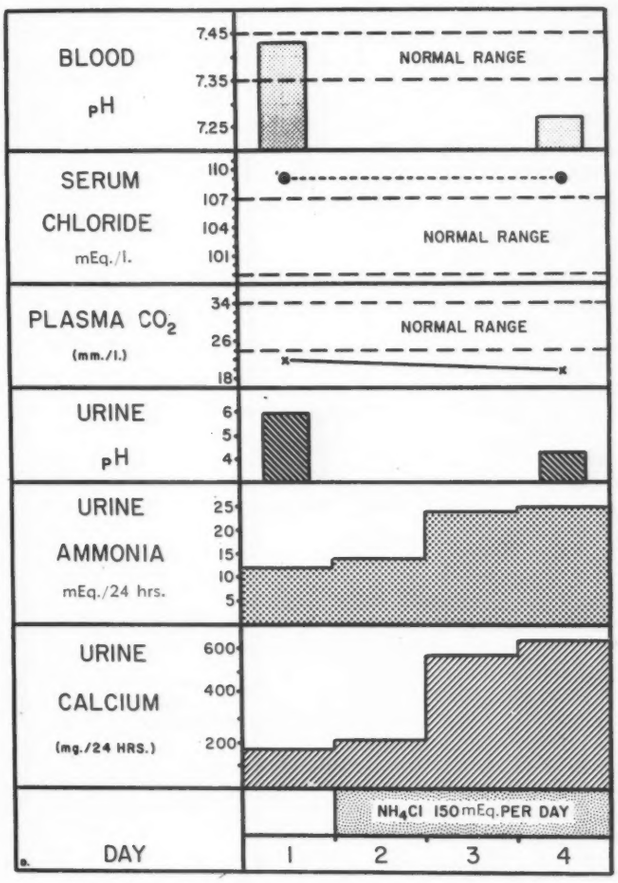

Fig. 2.—Biochemical findings in D.W., showing the presence of compensated hyperchloræmic acidosis and low basal urinary ammonia. Slight increase in urinary ammonia, marked hypercalciuria, and lowering of blood pH occurred during ammonium chloride loading.

ammonium chloride loading, though an appreciable rise occurred in the control subject.

Plasma CO_2 level was low in each of the children and fell slightly in each during the period of acid loading.

The urine was alkaline or slightly acid in each of the children and became strongly acid in all during ammonium chloride loading. Urinary ammonia in D.W. and J.W. was considerably less than that of the control before acid loading. In B.W. the basal value of 21 mEq. was close to the value obtained in the control subject. In all three children, however, little increase in urinary ammonia occurred with acid loading, in striking contrast to the profoundly increased values in the control subject.

Urinary calcium was not increased in any of the children during the resting phase. When ammonium chloride was administered, marked hypercalciuria with values over 600 mg. per day was noted in B.W. and D.W. This contrasts with the maximum value of 260 mg. in the control subject. In J.W., the 2½-year-old child, a maximum value of 288 mg. per day was reached.

DISCUSSION

The pathogenesis of renal tubular acidosis is not clearly known. The common clinical features are polyuria and polydipsia, the manifestations of renal calculi, pyelonephritis leading terminally to uræmia, and those which result from body calcium,

TABLE I.—ACID-BASE DATA IN CHILDREN AND CONTROL SUBJECT.

	BLOOD						*URINE*								
	pH		*Serum chloride (mEq./l.)*		*Plasma CO_2 (mm./l.)*		*pH*		*Ammonia (mEq./24 hrs.)*				*Calcium (mg./24 hrs.)*			
	Before acid load	*After acid load*	*Before acid load*	*After acid load*	*Before acid load*	*After acid load*	*Before acid load*	*After acid load*		*Ammonium chloride load*				*Ammonium chloride load*		
									Day 1	*Day 2*	*Day 3*	*Day 4*	*Day 1*	*Day 2*	*Day 3*	*Day 4*
B.W.	7.40	7.32	109	107	21	19	7.15	4.79	21	30	32	42	96	272	304	644
D.W.	7.41	7.27	111	110	22	18	5.90	4.79	12	14	24	25	176	215	570	623
J.W.	7.38	7.38	109	110	20	17	7.37	5.10	7	9	13	19	154	146	226	288
Control subject	7.37	7.36	103	109	26	23	5.75	5.25	23	51	80	83	96	146	224	260

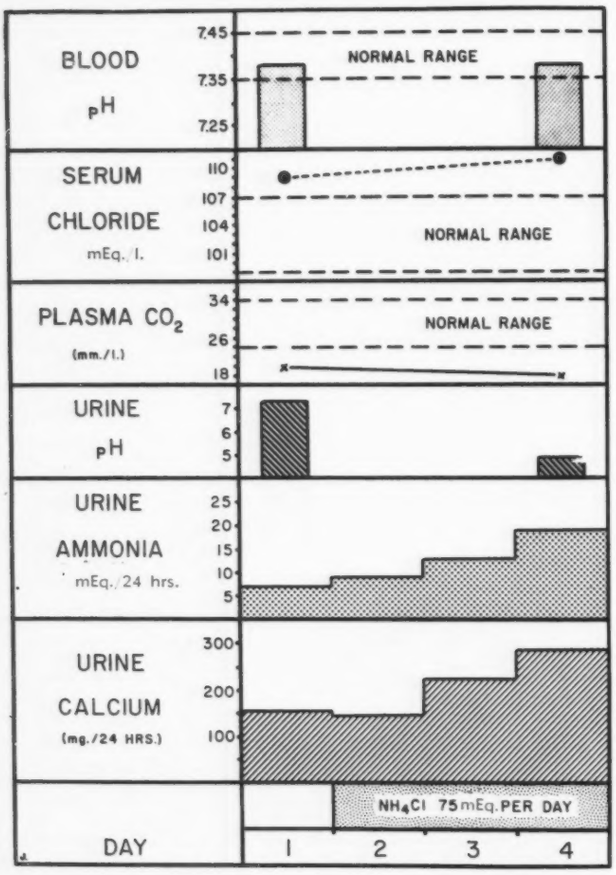

Fig. 3.—Biochemical findings in J.W., showing presence of compensated hyperchloræmic acidosis with alkaline urine and low basal urinary ammonia. Slight increase in urinary ammonia and moderate hypercalciuria occurred with ammonium chloride loading.

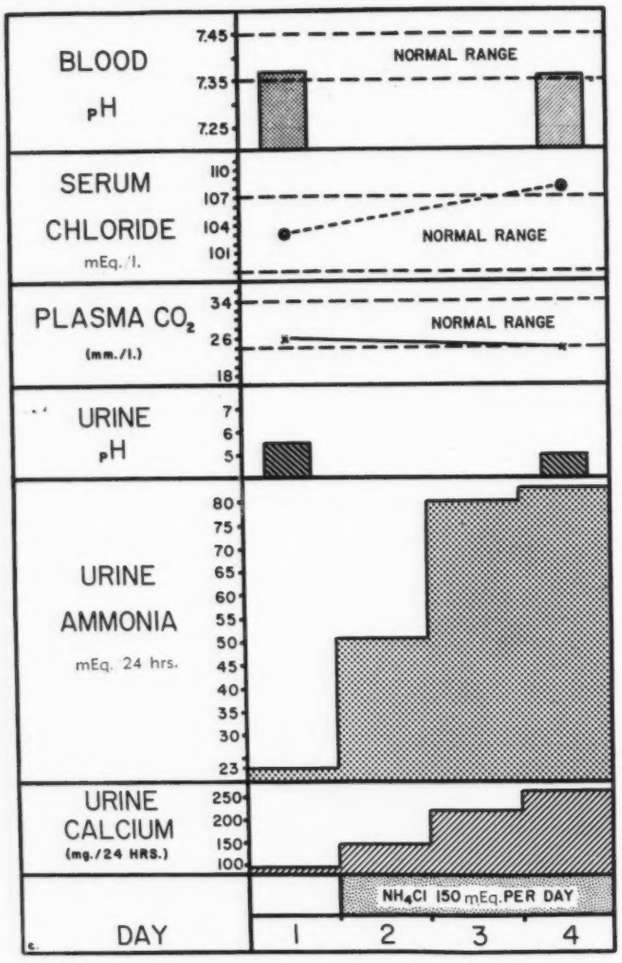

Fig. 4.—Biochemical findings in I.L., the normal subject, showing marked increase in urinary ammonia and slight hypercalciuria with ammonium chloride loading.

potassium and sodium depletion. The disturbance in acid-base metabolism is reflected in the hyperchloræmic acidosis with normal or low blood pH and alkaline urine. In addition, diminished ammonia excretion and impaired ability to increase ammonia excretion after acid loading have been reported.[3, 5, 6]

Clinical manifestations were entirely lacking in the subjects of this study. In each of the three, however, some biochemical manifestations of renal acidosis were evident. B.W. showed a compensated hyperchloræmic acidosis with slightly alkaline urine during the control period. While the urinary ammonia was normal at that time, there was little increase in excretion with ammonium chloride loading and pronounced hypercalciuria accompanied by lowering of blood pH (Fig. 2). D.W. showed similar findings, but, in addition, basal urinary ammonia excretion was normal (Fig. 3). In J.W. the findings were essentially the same, but lowering of blood pH did not occur and the hypercalciuria was not so pronounced (Fig. 3). These differences may have been due to the administration of a smaller acid load. Typical oxalate bodies seen in the urinary sediment of the mother[3] were numerous in the urinary sediment of J.W.

In each of the three subjects the urine became strongly acid with the ammonium chloride load. This suggests that renal tubular acidifying mechanisms were not as profoundly disturbed in the subjects of the present study as in those patients with the fully developed syndrome. Despite this, other renal responses to acid loading were impaired, and altered renal tubular control of acid-base metabolism was manifested by hyperchloræmic acidosis, alkaline urine and lowered urinary ammonia. These children therefore appear to have a "forme fruste" of the syndrome of renal tubular acidosis.

Alkaline urine with diminished urinary ammonia and hyperchloræmic acidosis are basic features of this syndrome. In the light of present-day knowledge of renal tubular acidifying mechanisms,[8] this association is best explained by the speculation that renal tubular hydrogen secretion is impaired. Furthermore, these acid-base abnormalities are identical with those changes that occur after administration of acetazolamide and sulfanilamide (renal tubular carbonic anhydrase inhibitors) to human subjects.[9, 10] Similarly, hypocitraturia occurs in this condition[11] and with acetazolamide therapy.[12] It would appear, therefore, that the central defect in this syndrome may be a qualitative or

quantitative abnormality of renal tubular carbonic anhydrase activity.*

While familial occurrence has been reported previously,[2] occurrence of manifestations of renal acidosis in all offspring of an adult patient has not. These observations suggest that the childhood and adult forms of idiopathic renal acidosis are one and the same condition.

It has been stated that this syndrome is never hereditary.[7] The findings herein presented suggest, in fact, that childhood and adult renal acidosis may be inherited diseases. Further observations on multiple generations of the families of individuals with this disorder are indicated to prove this. In the course of such investigations early manifestations of the disease will no doubt be detected so that treatment can be instituted to prevent the serious complications or fatal outcome.

SUMMARY

Observations are presented on the three apparently healthy offspring of an adult patient with idiopathic renal tubular acidosis.

All had some manifestations of the disorder, reflected by the presence of oxalate bodies in urinary sediment, the occurrence of hyperchloræmic acidosis, alkaline urine, diminished urinary ammonia and impaired renal compensations to an ammonium chloride load.

It is suggested that hereditary factors may be operative in the pathogenesis of this syndrome.

Childhood and adult renal acidosis appear to be one and the same disorder, in which the central defect may be an inborn error in renal tubular carbonic anhydrase metabolism.

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RÉSUMÉ

On présente les observations recueillies chez trois enfants apparemment en santé, issus d'un père atteint d'acidose tubulaire rénale idiopathique. A l'examen, tous les trois montrèrent des manifestations de ce trouble, révélées par la présence d'oxalates dans le culot urinaire, d'acidose hyperchlorémique, d'urine alcaline, de diminution de la formation ammoniacale dans l'urine et d'une compensation rénale insuffisante à une surcharge de chlorure d'ammonium. Les auteurs suggèrent que des facteurs héréditaires peuvent jouer un rôle dans la pathogénèse de ce syndrome. Il semble que l'acidose rénale chez l'enfant comme chez l'adulte soit identique et relève d'un défaut congénital du métabolisme de l'anhydrase carbonique tubulaire rénale.

APPENDIX

CASE 1

Clinical Findings

B.W., a 9-year-old boy with congenital spastic paraplegia, had a normal delivery and a birth weight of 6½ lb. Growth was normal and despite handicap he was able to walk satisfactorily. There was no history of enuresis, polyuria, polydipsia, or symptoms of renal lithiasis. Apart from signs of spastic paraplegia, physical examination was entirely normal.

Laboratory Findings

Urinalysis.—Albumin negative. Sugar negative; S.G. 1.026 (after 12 hours' dehydration). Sediment showed isolated calcium oxalate crystals. No pus cells were seen.

Hæmatological.—Hæmoglobin value 13.2 g./100 ml.; white blood cell count 8900, normal differential smear. Sedimentation rate 8 mm. per hour.

Biochemical values.—Non-protein nitrogen 36 mg./100 ml. Serum calcium 10.1 mg./100 ml. Serum phosphate 4.8 mg./100 ml. Alkaline phosphatase 22 King-Armstrong units.

Radiological.—Chest radiograph normal. Bone age normal. Excretory urogram: a double kidney and double ureter were present on the right.

Bacteriological.—Urine culture negative.

CASE 2

Clinical Findings

D.W., a 6-year-old boy, the second of the three children, had a normal birth and development. There were no symptoms referable to the urinary tract. General physical examination was entirely normal.

Laboratory Findings

Urinalysis.—Albumin negative. Sugar negative. Specific gravity 1.013 (after 12 hours' dehydration). Sediment: calcium phosphate and calcium oxalate crystals, the latter singly and in clumps. No pus cells were seen.

Hæmatological.—Hæmoglobin value 14.8 g./100 ml. White blood cell count 6200, normal differential. Sedimentation rate 0.

Biochemical.—Blood non-protein nitrogen 28 mg./100 ml. Serum calcium 10.1 mg./100 ml. Phosphate 5.2 mg./100 ml. Alkaline phosphatase 17.7 King-Armstrong units.

Radiological.—Chest radiograph normal. Bone age four years. Excretory urogram: normal.

Bacteriological.—Urine culture negative.

CASE 3

Clinical Findings

J.W., a 2½-year-old girl with normal birth and delivery, was well developed for her age. She did not

*The qualitatively normal response to acetazolamide in this disorder[1 13] suggests that renal carbonic anhydrase activity is not entirely lacking. This appears reasonable inasmuch as total absence of renal carbonic anhydrase probably would not be compatible with life.

drink undue amounts of water or urinate excessively. Physical examination was entirely negative.

Laboratory Findings

Urinalysis. – Albumin negative. Sugar negative. Specific gravity (random) 1.012. Sediment: calcium oxalate crystals and numerous oxalate bodies.[3] Five pus cells per high power field were observed in the urinary sediment.

Hæmatological.–Normal values.

Biochemical. – Blood non-protein nitrogen 27 mg./100 ml. Serum calcium 10.3 mg./100 ml. Serum phosphate 4.6 mg./100 ml. Alkaline phosphatase 19 King-Armstrong units.

Radiological.–Chest radiograph and excretory urogram normal.

Bacteriological.–Urine culture produced moderate growth of *E. coli.*

RECOGNITION AND MANAGEMENT OF DRUG-INDUCED EXTRA-PYRAMIDAL REACTIONS AND "PARADOXICAL" BEHAVIOURAL REACTIONS IN PSYCHIATRY*

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INTRODUCTION

NEUROLEPTIC (tranquillizing) drugs have a very widespread use, not only by psychiatrists but by the entire medical profession, in varied medical, surgical, and psychiatric conditions. This necessitates that all practitioners be familiar with both the drug-induced extrapyramidal reactions[4-16, 23] and the so-called "paradoxical" behavioural reactions[17-23] which can occur in response to the use of the neuroleptic and/or antidepressant drugs in psychiatry.

Although this information was originally prepared for circulation only among our residents, the realization that a paper, presenting in a fairly complete manner the differing aspects of this problem, was timely, prompted the presentation of this one. The recent report of Ehrlich[7] stated in part: "The recent occurrence . . . of a bizarre neurological syndrome due to phenothiazine tranquillizers and the lack of knowledge of physicians about this syndrome prompted this paper." Ehrlich's report led to the realization that publication of the present paper would be of some service.

Neuroleptic antidepressant drugs are given to reduce motor overactivity, to act on selected "target symptoms" that are part of psychotic and neurotic symptomatology; to control pain, to stop vomiting, or for a vast number of other reasons in medical practice. It will often happen that some patients, instead of having anxiety reduced, or overactivity controlled, demonstrate instead what has variously been called a "turbulent phase"[2] or a "paradoxical behavioural reaction".[3] In these cases the patients, instead of being calmed and having their motor overactivity reduced, became excited, agitated and more disturbed. We were the first to study and report the psychodynamic reasons for some of these reactions[17-23] which we have termed the "*psychologically determined* 'paradoxical' behavioural reactions". Before our studies, many believed that these types of reactions were organic in nature.[2, 3] We showed that there are two kinds of reactions: (a) those reactions that are *psychodynamically determined* and are caused by the patient's psychological reactions to the "pharmacological profile" of the drug, and (b) *neurologically determined* reactions, such as akathisia.

A. PSYCHODYNAMICALLY DETERMINED "PARADOXICAL" BEHAVIOURAL REACTIONS

In this first type, the typical pharmacological effect chemically removes or interferes with activities used by the patient as the major defences against unconscious underlying conflicts. These activities are therefore needed by the patient in order to feel relatively well. Such patients respond to the "chemical" interference with panic, agitation, increased despondency, paranoid reactions, distortions of body image, increased withdrawal, increased agitation, or markedly enhanced anxiety.[17-19, 21, 22] All of these occurred because of the psychological meaning to the patient of the physiological effects of the drug.

Azima *et al.*[1] offered a psychodynamic visualization of the "turbulent" phase seen with reserpine in chronic schizophrenic subjects, as a "manic" phase of schizophrenia. This visualization is one that parallels the theoretical constructs previously reported by us.[17-22]

Our findings will now be summarized. Drugs can threaten the following categories of patients:[17-22]

1. Male patients who mask profound doubts about their masculinity by using social outgoingness and intellectual and motor activity as the major defence against underlying unconscious feminine identification and strivings towards passivity: To these patients, activity = masculinity, passivity = femininity. Drugs which remove motor overactivity can, for this reason, cause markedly

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increased anxiety and agitation instead of reducing them, as well as produce changes of body image and further neurotic or psychotic disturbance in the thought processes.

2. Cases of marked withdrawal without anxiety or conflict about this: Further withdrawal and apathy may be caused in some patients by further reduction in the energy available for muscular and externalized activities.

3. In depressive reactions, largely those without agitation or marked obvious anxiety, neuroleptic drugs can increase withdrawal and apathy in the selected patient by further reducing the amount of available energy. These drugs can, by reducing such patients' capacity for exteriorizing any emotion, and lessening affective responses in their interpersonal relations, increase the patients' feelings of personal worthlessness, and thus increase their level of depression. In short, the drugs act as agents which cause further introjection of already intolerable aggression. They further limit, by reducing available energy for bodily activities and interpersonal relations, those activities which represent some exteriorization of such aggression.

4. Patients with marked preoccupations over body image, or ruminations over body integrity: In this category are patients with marked concern about their ability to control their bodies, as well as obsessive-compulsive or phobic patients with obsessive ruminations over the possibility of disease and impaired bodily health. Patients with borderline states are also often found in this category.

Drugs with powerful pharmacological effects are interpreted by some of these patients as decreasing their ability to control their own bodies. Some patients with such problems interpret the side effects as markedly threatening. These side effects suggest bodily impairment, and bring closer to the surface their fears of retribution for "sins" and guilt in terms of bodily disease, as though it is expressed in a living tableau before their very eyes.

The patients with borderline states often have poorly formed body images, and interpret the physiological effects of the drug as permanent changes in their body, or as putting their body in the doctor's power. With many of these patients the side effects of a drug are perceived as a further bodily impairment.

Such patients often develop the feeling that their bodies are changed and are no longer their own. They show projective thinking and paranoid mechanisms, and often show marked somatic preoccupations (ruminations) of a greater degree than the side effects might warrant, as well as markedly increased anxiety and depersonalization.

5. Some patients in the early stages of a chronic brain syndrome with preoccupations over bodily function and bodily health are sometimes frightened by side effects which they do not understand and cannot account for.

6. Patients to whom the administration of any drug is interpreted as an unwelcome assault or seduction: They may interpret this as either a heterosexual assault or a homosexual assault. They often wonder why the doctor wants them to "lie in bed" under the effects of the drug, or why he is "weakening" them, making them "tired", "dizzy", etc.

7. All patients in whom the presence of marked secondary gain from their illness[25] makes the removal of "target symptoms" improbable or impossible: Some of these patients show bizarre responses to drugs, since they do not wish to lose symptoms for reasons which have nothing to do with the drug action (desire for pension, compensation, etc.).

8. Certain patients whose cultural and familial background has conditioned them with attitudes toward life that can best be termed as "magical": Here, family beliefs in the mystic and the occult have resulted in the patient's inhabiting the world with "magical" forces. These are invoked by the patient in reference to the drug action. Such patients feel that the drug has magical qualities, puts "goodness" into them, and drives "badness" out of them.[27] Sometimes, however, the drug effects may be interpreted as putting "badness" into them, and then doing greater harm than good. When it is the latter, they endow the drug and the doctor with the intention, or suspicion, of performing "bad magic", and often equate the physiological action, or side effects, in modifying bodily function and the energy available for external activities with this. They equate symbolically linked material, which has specific, unconscious meaning to them, with the dramatic pharmacological action of the drug. When they feel that this is "bad medicine" or "bad magic", such patients can become markedly disturbed along the lines already described.

9. Panic reactions: Many patients who develop panic reactions after the first few days of drug administration, with intense anxiety, agitation, sweating, palpitation, and dilatation of pupils, or who complain bitterly about the drug, may do this on any and all the drugs tested, and such patients uniformly consider the medication, regardless of its type, as putting them in the "doctor's power". To such patients this is threatening for varying reasons.

Nosology

The nosological picture produced with the psychodynamically determined "paradoxical" behavioural reactions varies from patient to patient, and depends on the exaggeration of the pre-existing personality of the patient and on the patient's total situation. We have mentioned some of the more common reactions.

If the practitioner keeps in mind the possibility of a worsening in the patient's state in response to

the patient's feelings about the physiological effects of the drug, and remembers that this can be linked to the meaning that the drug action has had for the patient, he will find these reactions easier to diagnose. This adverse psychological meaning always exists in terms of the patient's feeling that he is being changed for the worse, and that the drug is therefore harmful. Similar reactions have been seen with the antidepressant drugs.[28-30]

Such psychodynamically determined "paradoxical" behavioural reactions can occur with any drug and are not dependent on the chemical nature of the radicals concerned. They are dependent on the *psychological meaning* to the individual patient of the pharmacological action of the drug as it is perceived by the patient. From the patient's viewpoint, all the external factors that may influence this perception of the drug effect as either good or bad are operative. This includes factors in the total situation such as the patient's relationship with the doctor, the reality situation including all the complexities of it, the transference feelings, and the way the patient perceives his doctor's attitude towards him, this is to say, the way the physician's unconscious attitudes toward the patient are perceived by the patient. The social setting or milieu, and the many variables that operate in the milieu to give it the character of being positive (i.e. good) or negative (i.e. bad) for the patient, are important here.[31, 32] For it is the totality of all these factors which determines whether a patient will or will not have such reactions.

If understood in the light of the above, there is nothing "paradoxical" about the psychodynamically determined behavioural reactions.

B. Drug-induced or Neurologically Determined Extrapyramidal Reactions

In contradistinction to the psychodynamically (psychologically) determined "paradoxical" behavioural responses just described, a specific group of *neurological* pharmacologically induced side effects will now be considered.

Freyhan has divided the phenothiazines into what he has called the "chlorpromazine model" phenothiazines and the "prochlorperazine-model" phenothiazines.[8, 9] The former have in common three carbons in a straight chain, while the latter have the piperazine radical at the end of the three-carbon straight chain.

Generally speaking, and this is *by no means absolute*, the chlorpromazine-model compounds cause greater peripheral effects (such as vascular changes and blood pressure changes) and a greater incidence of drowsiness, and often seem to "flatten" a patient to a great degree. The prochlorpromazine-model compounds show fewer blood pressure and other peripheral reactions, and allow the patient to be up and about to a greater degree, but cause a higher degree of extrapyramidal syndromes.

Although the drug-induced extrapyramidal side effects can occur with any of the phenothiazines and with the rauwolfia alkaloids (reserpine, etc.), they occur particularly with the prochlorperazine-model phenothiazines. It must be emphasized that in some patients they occur with the use of other phenothiazines, rauwolfia compounds, and chemically different compounds in use as tranquillizers, as well as with some of the "anti-depressant" or stimulant variety. Examples of drugs which are particularly apt to cause extrapyramidal side effects are prochlorperazine, perphenazine, trifluoperazine, and many other piperazine radical phenothiazines.

Extrapyramidal Reactions

The drug-induced extrapyramidal-like syndromes are no different from those clinically seen following naturally occurring disease, such as parkinsonism, and the postencephalitic varieties of basal ganglia disturbances. In the naturally occurring diseases, these reactions tend to be permanent. In the drug-induced reactions they are completely reversible, and are primarly *neurologically* (not psychodynamically) determined. They are caused by the action of the neuroleptic drug on subcortical structures, mainly the basal ganglia and its relay connections.

The drug-induced extrapyramidal side effects, including the behavioural components of these, can go from zero to infinity in degree. Thus, they can be minimal and mild as to symptomatology and discomfort in the patient, or they can be extremely troublesome, showing a fully developed tableau.

Among these reactions are:

1. *Parkinsonism or parkinsonian-like reactions.*—These reactions are characterized by gait and postural abnormalities that include varying degrees of rigidity, tremor, and salivation. Their incidence and time of occurrence vary with the drug and the dosage used. As a generalization, the symptoms may be said to occur very early in drug administration, usually during the first eight days, often in the first three. The main exception to this is their occurrence, in patients already receiving the drug for some time, after a drastic increase in dose, or after administration of the drug intramuscularly or parenterally. Muscular rigidity, a mask-like facies, an inability to move the head and trunk except in a block, in some cases salivation and drooling—all can be part of the picture.

A general increase in muscle tonus and rigidity, clinically perceivable, but not reaching the level of a full-blown parkinsonian syndrome as outlined above, is seen in a number of cases, and is probably more common than the full-blown syndrome. This may be considered as a variant of the fully developed parkinsonian syndrome, from which it varies only in the question of degree.

2. *Dyskinetic and dystonic syndromes.*—These are characterized by spasms or cramps, either generalized or isolated to several muscle groups. They can again vary in severity from the very mild to the extreme torsion and spasms. Muscle spasms are often seen. Muscle cramps, either generalized

or isolated to several muscle groups, are very common. Cramp-like pain and spasm is often seen in the legs, frequently in the calf muscles, the arms, and the muscles of the back of the neck or trunk. Syndromes involving the face and neck are among the commonest seen. Trismus is common, but much more frequently there occurs some tightness of the jaw muscles, with difficulty in masticating or in opening the mouth, though not of sufficient degree to warrant classifying it as trismus. Torticollis as a separate clinical syndrome is not uncommon. Mandibular tics, speech and swallowing difficulties due to hypertonicity in various regional muscles, oculogyric spasms, torsion spasms, tonic contractions and myoclonic twitches of the lateral or bilateral muscle groups are often seen in addition to the more frequently described reactions already mentioned.[8]

In the odd case the hypertonicity of some muscle groups becomes very marked, and can produce opisthotonus, trismus, risus sardonicus, and forced protrusion of the tongue outside the mouth. Such cases have been mistaken for tetanus by physicians who are not familar with the above syndrome, or who have not been informed that the patient has been taking a neuroleptic drug.[7] These somewhat dramatic manifestations are not of serious import, and they are rapidly reversible when treated properly. The dyskinetic or dystonic syndromes tend to appear in the first eight days of drug administration, and most commonly in the first three. They occur with greater frequency if the initial doses are high.

It is important to point out that these manifestations appear, fade, and return often with a seizure-like rhythm. Sometimes their appearance is accompanied by autonomic signs such as profuse sweating, pallor, and occasionally fever. Although these manifestations can often be dramatic in their intensity, consciousness is never impaired, the patients remaining fully alert.[6, 8, 9]

3. *The excito-motor syndrome.*—A variant of the dyskinetic syndrome, it was originally described in the neurological literature. This syndrome is occasionally drug-induced.

Characterized by many aspects of the dyskinetic syndromes already described above, but in their most dramatic form, this syndrome was first observed by Marie[15] and Levy[15, 16] in the preparkinsonian phase of the postencephalitic disorder that followed Von Economo's sleeping sickness, i.e., as an early sequel. They described a series of abnormal movements and psychomotor difficulties characterized by myoclonic twitchings and jerks, tonic spasms, and choreiform rhythmic movements, to which shivering of the type seen in response to cold was added. Accompanying this was a peculiar modification of the emotional state in which the patient, although maintaining full consciousness, seemed to centre his attention more upon himself, and seemed intensely preoccupied with a diffuse, unpleasant type of sensation which he could not at the time easily ignore. One can speculate about a possible link with the type of sensation seen in thalamic disorders. During the excito-motor syndrome, or as a special variant of it, an affliction of the facial, lingual, and masticating muscles is sometimes seen.[6] Trismus, which the patient can temporarily abolish when influenced to do so, or by a voluntary effort, but which immediately recurs, is part of this syndrome. This trismus can also temporarily disappear on passive opening of the mouth, and can alternate with spasms in which the mouth is forced open and left in this position. The lips may perform asymmetrical grimaces accompanied by movements of suction or protrusion of the lips. The tongue is often involuntarily thrust outside the mouth beyond the limits of the teeth, but will often be retracted when its tip is lightly touched, or on the suggestion that it be withdrawn. This lasts for only a short time, after which it again protrudes. These disturbances in the tonus of the lingual and masticatory apparatus interfere with mastication and speech, although swallowing difficulties are not too often described. This sub-syndrome seems identical to that described by Kulenkampf[14] as the "oral syndrome" that can occur with chlorpromazine.

Oculogyric crises have been seen, as well as dystonic movements, torsions, torsion spasms, with localized or generalized choreo-athetose movements, torticollises and "bradykinetic"[6] movements. The latter are slow movements of large amplitude which are repeated with a slow rhythm. Some patients have spasms involving much of the body with considerable torsion and writhing.

This rather dramatic and alarming syndrome should be considered as a variant of the dyskinetic reactions.

Akathisia

Following the report of Von Economo's sleeping sickness, Sicard[26] described a syndrome of restlessness, agitation, and insomnia occurring in the preparkinsonian phase of postencephalitic disturbances. He described "tasikinesia" as a "tendency to movement", and "akathisia" as the "impossibility of sitting down". Contained under these two names is a special form with agitation and insomnia, the need to move, and the inability to remain seated.

Later similar states were drug-induced and reported with use of reserpine, and particularly with prochlorperazine. They are particularly frequent with the prochlorperazine-model phenothiazines.

The above syndrome is today generally referred to as "akathisia". This is a specific syndrome characterized by a feeling of "inner restlessness", "motor restlessness" or "inner drivenness", and the features already mentioned. Again, it can exist in all degrees from zero to infinity. Some patients feel that they must move and keep pacing, while others feel that they must move their feet. Some authors have called it the "restless feet syndrome" as a result

of this. Inaction becomes unbearable for these patients. When the syndrome exists in a mild form, the patient may describe that he has the feeling of "being driven", with an increased feeling of tension; sometimes with a feeling of a "pulling" or "drawing" in the extremities (chiefly in the legs). In the more fully developed syndrome, patients pace back and forth; they cannot read, play, or sleep. In severe cases, patients may appear to be continually agitated.

All of the abovementioned reactions are drug-induced, are *neurologically* determined, and are caused by the effect of the drug on the basal ganglia and their connections.

Incidence of the Extrapyramidal Drug-induced, Neurologically Determined Reactions

The incidence depends on the particular drug and on the *dosage used*. The incidence with prochlorperazine-model phenothiazines can vary from 40% to 100% of the patients receiving the drug. As a general rule, parkinsonism exceeds the others in frequency, with dyskinesia second, and akathisia third. It must be remembered, however, that the incidence varies with the dose used. On low doses you may see none or very few of these reactions, and there is also evidence for sex-linked differences. Parkinsonism seems to be approximately twice as frequent in women. Akathisia, and particularly the dyskinetic reactions, tend to prevail in men. Some authors feel that parkinsonism and dyskinesia are relatively independent reactions. Whereas akathisia is often considered to be one of the features of parkinsonism,[8, 24] dyskinetic reactions seem to occur in the very early phases of the drug administration, and are considered by some to represent the sensitivity of the subcortical structures to the drug effect, which disappears as the patient adapts to it.[8]

DIFFERENTIAL DIAGNOSIS

In earlier work we described in detail what we called the "psychodynamically determined behavioural reactions". The causes of these have been enumerated and physicians who keep these causes in mind will find it easier to recognize those patients who, for psychological reasons, respond to drug effects with increased anxiety, agitation, complaints, body image changes, preoccupation with disease or side effects, or increased psychosis. These occur for psychodynamically determined reasons.

These reactions occur with all drugs, not just mainly with the prochlorperazine-model phenothiazines. They must be carefully differentiated from the *neurologically* determined extrapyramidal reactions described above. The *psychodynamically* determined reactions occur for psychological reasons because the drug interferes with defences necessary for the integrity of the patient, while the *neurologically* determined reactions occur because of the pharmacological effects on subcortical, and particularly basal ganglia regions.

With the occurrence of the neurologically determined reactions (parkinsonism; dyskinetic reactions, including the psychomotor syndrome; akathisia), some patients will develop *secondary* psychodynamically determined behavioural reactions as previously described, in response to the psychological meaning to them of the neurologically determined side effects. By this I mean that a minority of patients, in response to the extrapyramidal side effects, become panicky, fearful, and agitated, because they say in essence, "What, Doctor, are you doing to me? what are you doing to my body, and why?" They then react along the lines enumerated for the psychological reactions in the above discussion. Here then one has a *primary* neurological disturbance, and a *secondary* psychological disturbance in response to the meaning the neurological symptoms have for him.

Thus, it is important for all physicians to keep these three points in mind: There are—

1. Psychodynamically determined so-called "paradoxical" behavioural reactions.
2. Neurologically determined reactions such as parkinsonism, dyskinetic reactions, and akathisia.
3. Neurologically determined reactions, on to which a secondary panic, or other psychodynamically determined reactions can be grafted.

MANAGEMENT

It is first necessary to determine from which of these reactions the patient is suffering. If he seems to show panic, or other psychologically determined reactions, the patient should receive an explanation of what is occurring, and the reasons for it. If side effects are triggering some of his difficulties, provided that his clinical state permits it, the dose level of the drug can be reduced, thereby limiting side effects. Explanations and reassurrances often help many of these patients face physiological effects that are psychologically troublesome. For others, when the psychodynamically determined reactions occur, the only reasonable action is to discontinue the drug. Many such patients feel much better when this is done.

When using *any* drug, but particularly the prochlorperazine-model phenothiazines, one should keep in mind the necessity of beginning with as low a dose as is clinically feasible, for the first two or three days, and then increasing the dosage slowly if necessary. This principle should apply, except in an emergency, when one can give these drugs in high doses intramuscularly or orally, accepting the risk of the extrapyramidal reactions, but being quite prepared to manage them by appropriate measures if they arise. If the drug is begun in fairly low doses for the first two or three days, and then increased slowly, the incidence of extrapyramidal reactions is eliminated or reduced. Once the patient is receiving the drug, any sudden change to higher dosage may also cause one of

these side effects. They should not be feared by physicians, who should be prepared to increase the dose as necessary, and to manage the extrapyramidal reactions should they occur.

1. *Management of dyskinetic reactions*[8, 24].—The management of the dyskinetic reaction depends on its severity. If it is mild and not too disturbing, omitting one or two doses of the drug, or substituting oral for intramuscular administration, often suffices. A dramatic onset of severe dyskinesia, however, should be treated promptly. If it occurs in the daytime, in a severe case an intravenous injection of a stimulant such as caffeine, sodium benzyl, 7.5 grains intravenously, often abolishes all symptoms within approximately half an hour to an hour. If this response is not adequate, the injection should be repeated. If the patient is intensely agitated, and especially if this occurs at night, sodium phenobarbital, 2 grains, can be administered intravenously, and this can be repeated if the symptoms persist until the patient sleeps. An antiparkinsonian compound,[8, 24] such as trihexyphenidyl (Artane), 1 mg. or 2 mg. dosage, or procyclidine (Kemadrin), 10 mg. or 20 mg., or benztropine (Cogentin), is often highly effective. It is usually necessary to continue the administration of trihexyphenidyl (1 mg.) or procyclidine (5 to 10 mg.) thereafter concomitantly with each dose of the drug given. For some patients the administration of the antiparkinsonian medication twice daily will suffice. On the day of a severe dyskinetic reaction, additional doses of the drug may be omitted. The regular schedule can usually be resumed the next day. Use of caffeine or phenobarbitone may not be necessary at the onset, if the dyskinetic reaction develops gradually. Then, the antiparkinsonian medication alone suffices in most cases. Reduction of the dosage of the phenothiazine drug and concomitant administration of the antiparkinsonian compound usually suffices to alleviate the disturbance in a few hours. The severe dyskinetic reactions appear more frightening than they really are. The patient is usually in no danger, although the manifestations are often dramatic. If he is treated with the above techniques, there should be no serious or irreversible effects, the patient recovering very rapidly and resuming his usual behaviour as soon as the reaction subsides. The disturbance is a functional one originating in the midbrain, so that treatment depends entirely on the drugs and on the effects of the central action of these drugs. Fright or panic reactions, or anxiety about this, should be interpreted to the patient as caused by the drug. Most patients are not frightened or shocked by this if proper explanations are offered and the condition is remedied by appropriate means.

2. *Parkinsonian reactions.*—These include muscular rigidity, gait and postural abnormalities, varying degrees of rigidity, tremor, and salivation described above. Treatment should consist of reduction of the dosage of the drug and, in addition, patients should be given an antiparkinsonian agent such as trihexyphenidyl (Artane), benztropine (Cogentin), or procyclidine (Kemadrin). This can be given concomitantly with each dose of the drug as soon as definite evidence of parkinsonism develops. One should, however, in all cases adjust the drug to keep the level of the antiparkinsonian compound as low as is necessary. Routine administration of antiparkinsonian componds should be discontinued after the parkinsonian reaction has disappeared.

3. *Akathisia.*—Management of akathisia may at times be somewhat more difficult. First and foremost, drug dosage should be reduced. Trihexyphenidyl (Artane) and benztropine (Cogentin) are effective in the dosage mentioned. Procyclidine (Kemadrin) in daily dosage of 10-30 mg. is reasonably effective. The dosage level should be reduced, barbiturates can be added in appropriate doses at night, and other tranquillizers of a non-prochlorperazine-model type may have to be added to enable the patient to sleep, and during the daytime by the use of trihexyphenidyl and benztropine or procyclidine patients can usually be controlled.

Using these guides, no physician should fear the use of prochlorperazine-model phenothiazines because of extrapyramidal reactions. The extrapyramidal reactions can be managed, and it is the duty of the physician to learn to do so when necessary.

Implicit in all the above is the indication that no drug will be given except when necessary, that no tranquillizer, powerful neuroleptic, or stimulant drug will be given when a simpler drug may suffice, and that the dosage level of the drug used will be appropriate to the symptomatology or target symptom that one is attempting to control.

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RÉSUMÉ

Certains malades mentaux recevant des neuroleptiques réagissent d'une façon paradoxale aux médicaments qui devraient les calmer et résoudre leur hyperactivité. Cette réaction d'angoisse prend la forme d'excitation, d'agitation et de désordre. La phase turbulante peut se manifester chez les hommes qui ne sont pas tout à fait convaincus de leur masculinité, chez les malades déjà profondément retirés en eux mêmes, dans les cas de dépression par réaction, chez ceux que la représentation corporelle préoccupe de manière exagérée et enfin chez les malades atteints d'un début de syndrome cérébral chronique. Il faut également inclure tous ceux qui se méfient des médicaments en général et qui soupçonnent le thérapeute de vouloir les affaiblir, voire les empoisonner, ceux qui misent sur leurs symptômes dans l'intention d'en retirer un avantage secondaire et les autres qui sont portés à une réaction panique quel que soit le médicament employé. En plus des réactions d'ordre psychodynamique, ces médicaments peuvent également produire des symptômes neurologiques d'ordre organique. Ce sont le plus souvent des symptômes extra-pyramidaux comme ceux que l'on observe dans la maladie de Parkinson; ils comprennent la rigidité, la trémulation et le ptyalisme, la dyskinésie et le syndrome excitomoteur ainsi que l'akathisie. La conduite du traitement repose d'abord sur un bon diagnostic. On doit ensuite s'efforcer de rassurer le malade et de lui expliquer la cause des troubles qu'il ressent. Il importe d'administrer ces médicaments de façon judicieuse d'abord à petite dose quitte à augmenter la posologie graduellement. Les réactions dyskinésiques cèdent souvent à la médication antiparkinsonienne mais il est quelquefois nécessaire de diminuer la dose de neuroleptiques.

Case Reports

POLYCYTHÆMIA AND FIBROMYOMA OF THE UTERUS

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ERYTHROCYTOSIS in association with benign and malignant tumours has been known for 30 years. The first observations were reported in 1929 by Oppenheimer[1] and Castex.[2] The first case was a cerebellar medulloblastoma, and the second a tumour of the hypophyseal region. These were followed by reports of neurological tumours,[3-9] hypernephromas[10-12] and ductless gland tumours[5, 13] accompanying polycythæmia, and in 1953, Thomson and Marson[14] reported a case of polycythæmia secondary to or associated with fibromyoma of the uterus.

We would like to report a case of fibromyoma of the uterus accompanied by polycythæmia. To date, five similar cases have been reported.[15-18]

Mrs. A.G., aged 57, was admitted to hospital in February 1955, because of metrorrhagia. Her menopause was four years previous. She had an enormous hard mass, well limited, in the abdomen. The mass originated in the pelvis and could be delineated as high as the xiphoid process, taking the form of butterfly wings.

Red blood cell count was 6,900,000; hæmoglobin value 20.4 g. %; other hæmatological and biochemical studies were normal. The patient was not operated upon but was sent home temporarily to permit healing of an eczematous dermatitis.

The patient returned in May 1955. The R.B.C. numbered 8,100,000; Hb. 19.5 g. % and hæmatocrit value 78%. Total blood volume determined by the Evans blue method was 8955 ml., and red cell volume 6985. Reticulocytes were 1.8%. Material from sternal puncture revealed intense normoblastic hyperplasia only. A Van Slyke test value was 38.6% of the normal clearance. She had congestion of the face and mucous membranes, dyspnœa on exertion, hypertrophy of the left ventricle, the enormous abdominal mass described above, and œdema of both ankles. The rest of the examination and the analyses were unremarkable. The patient underwent a few venesections to prepare her for operation and was again allowed a temporary discharge.

At her third admission in August 1955, physical examination gave the same findings. R.B.C. was 5,300,000 (due to several venesections); Hb. value, 13.3 g. % and hæmatocrit 50. Blood sugar level was 152 mg. %; the oral glucose tolerance test was slightly positive. Blood urea nitrogen value was 21.5 mg. %. Protein and electrolyte values were within normal ranges, and the result of the Van Slyke test was 44.5%. Urine was normal and P.S.P. value was 31.5%.

On August 22, 1955, the patient underwent total hysterectomy and bilateral salpingo-oophorectomy. At operation a large bilobar uterine intra-ligamentary fibroma was observed arising behind the posterior peritoneum, from the floor of the pelvis up to the liver. The pathological report stated: large uterine fibromyoma with partial sclerosis, weighing 4700 g.

The day after operation the R.B.C. was 4,980,000; Hb. value 12.9 g. % and hæmatocrit 47%.

On September 7, the day before discharge, the Hb. value was 71.6%, hæmatocrit 41%, and R.B.C. 4,200,000.

At a later admission in October 1957, for the control of diabetes, the R.B.C. was 4,150,000; Hb. value 15.3 g. %, and hæmatocrit 48.5%.

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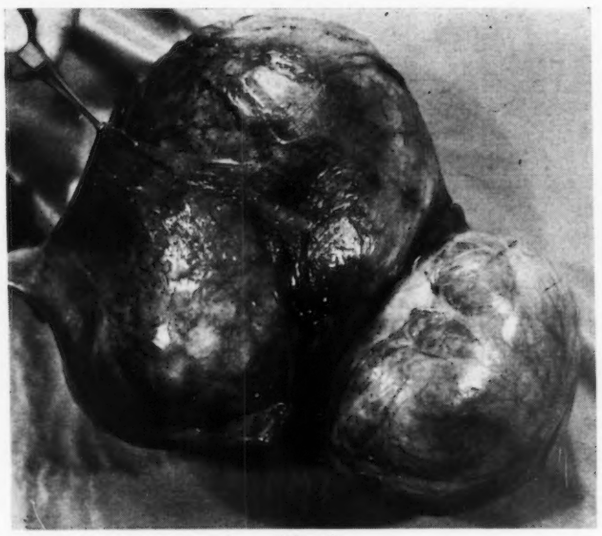

Fig. 1

DISCUSSION

In the observations reported to date by other workers, surgical removal of the uterine fibromyomas was followed by return to normal of the red cell volume. One of the patients was at the beginning of the menopause, another was postmenopausal (like our patient), and three were premenopausal. Their ages varied between 29 and 57. Hypertension was present in three. None had splenomegaly.

Myomatous polycythæmia[17] is easily differentiated from polycythæmia vera, clinically, hæmatologically and therapeutically, by the absence of splenomegaly, of thrombotic or hæmorrhagic tendency, of leukocytosis, and by normal reticulocytosis and definite postoperative improvement. It does not seem to resemble polycythæmia secondary to anoxæmia (sulfhæmoglobinæmia, methæmoglobinæmia, congenital cardiac disease, chronic bronchopneumonia, peripheral arteriovenous shunts, Ayerza's syndrome and high-altitude polycythæmia).

None of these patients had signs of Cushing's syndrome or of intracranial tumour. The pathogenesis of myomatous polycythæmia is still obscure. Horwitz and McKelway[16] suggested that arteriovenous shunts in the uterine tumour were an etiological factor. In the seven reported cases of polycythæmia associated with uterine fibromyomas, the theory of arteriovenous shunts in the tumoural mass could not be confirmed by histopathological examination.

A second theory is that a humoral factor originates in the uterine tumour and stimulates the normoblastic series of the bone marrow. Some workers[18] reject this theory because return to normal of the blood count, instead of occurring around the seventh postoperative day, would take several weeks after surgery. We think that this theory is acceptable and that the rapid return of the blood volume to normal does not eliminate it. It should be recalled that the blood volume of these patients was reduced to normal, even before the surgical procedure, by repeated venesections, and that bleeding during surgery would cause further decrease in blood volume. On the other hand, excision of the fibromyoma which causes sudden removal of the hypothetical humoral factor would be followed by rapid disappearance of the normoblastic hyperplasia of the bone marrow. If this hyperplasia lasted for only a few days after hysterectomy, it would not lead to recurrence of polycythæmia.

To reach sounder conclusions, the erythropoietic activity of the serum or plasma of these patients should be tested in laboratory animals. This could establish the presence or absence of a hæmopoietic humoral factor in patients with myomatous polycythæmia.

SUMMARY

A case of uterine fibromyoma associated with polycythæmia in a 57-year-old woman has been reported. This is the seventh such case reported in the medical literature. Hysterectomy, performed after several venesections, resulted in a complete cure of the polycythæmia.

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RÉSUMÉ

Un septième cas de polycythémie associé à un volumineux fibromyome utérin, chez une patiente de cinquante sept ans, a été rapporté. L'hystérectomie pratiquée après des phlébotomies répétées, a été suivie d'une rémission complète et permanente de la polycythémie.

USE OF SOAP BY PATIENTS WITH ECZEMA

A study conducted by Cleveland dermatologists indicated that the use of ordinary toilet soap for routine bathing and hand-washing has no deleterious effects on a variety of eczematous skin rashes during treatment by the usual therapeutic measures. The adverse influence of soap on such skin rashes, which has been claimed so frequently in the past, was not substantiated by these investigators, who concluded that it appeared rational to permit the use of soap in the management of patients with eczema without fear of unfavourable effects on the course of their disease.—*A.M.A.* News Release.

SURGICAL TREATMENT OF PARAPARESIS IN A PATIENT WITH ARRESTED TUBERCULOUS MENINGITIS*

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INTRODUCTION

Survival of patients with adequately treated tuberculous meningitis has become increasingly common in recent years. However, the management of some of the residual neurological deficits in such patients has not yet been clearly delineated, since this is a problem of relatively recent vintage, and with relatively little clinical material upon which a decision regarding such management may be based. Hence the possible value of the present case report. When the author was confronted with the necessity of deciding whether or not to operate on this particular patient, he was unable to obtain any definite help, based either on personal experience or hearsay or knowledge of pertinent literature, from quite a few members of the Canadian Neurological Society, whom he had the opportunity of consulting during a meeting of the Society taking place at that time.

Chortis,[1] reporting on therapy of transverse myelitis occurring during tuberculous meningitis, stated that such transverse myelitis might occur in a considerable number of cases of tuberculous meningitis. However, in his opinion the myelitis appeared in cases of prolonged and incomplete therapy of tuberculous meningitis, and mainly when intraspinal injections of streptomycin had been administered for a long time. He did not mention the possibility of surgical treatment of such cases, but he did claim some benefit from administration by mouth of a preparation described as "Sulfone J.51", especially if this drug were given before complete degeneration and necrosis of one or more spinal segments had occurred.

The subject of this case report was a 15-year-old high-school girl, D.M. She was first referred to the Saint John General Hospital on December 4, 1957. There was a history of an "influenza-like" illness, beginning about five weeks previously. After a week or so the girl had seemed to improve, but anorexia continued, along with intermittent headache and nausea. On November 25, 1957, the patient was admitted to the Hôtel-Dieu Hospital in Perth, N.B., and treatment with penicillin and chloramphenicol was instituted. However, the patient showed increasing drowsiness and disorientation, while her temperature varied between 98° and 101° F. She was referred to the neurosurgical service of the Saint John General Hospital as a diagnostic intracranial problem.

On admission, on December 4, the patient was not normally conscious; at times she was difficult to reach verbally; at other times she did respond verbally, but there was evidence of disorientation in time and place; she yawned frequently; some degree of nominal aphasia could also be demonstrated. The vital signs were satisfactory, though the blood pressure and pulse rate were quite variable. The neck was markedly stiff, and when not disturbed the girl tended to "curl up" on her right side. The pupils were unequal, the left larger than the right, and responded more actively to accommodation than to direct light stimulation. The temporal half of the left optic disc appeared somewhat pale; the nasal side of the right optic disc was slightly elevated, and the retinal veins on this side seemed abnormally full. In other respects the neurological findings were either within normal limits or impossible to evaluate accurately, owing to inadequate co-operation on the part of the patient.

A lumbar puncture was performed. The initial cerebrospinal fluid (C.S.F.) pressure was 270 mm. of water. The fluid was xanthochromic and cloudy, with a white cell count of 100 polymorphonuclear cells and 205 lymphocytes per c.mm.; the protein content was 1800 mg. %; the chlorides were 597 mg. % and the sugar 36 mg. %. A second lumbar puncture yielded essentially the same findings. Both specimens showed pellicle formation, but the initial careful searches for organisms by smear and general culture and by pellicle staining were negative. However, a tuberculin patch test was positive, and the presumptive diagnosis of tuberculous meningitis was subsequently substantiated by animal inoculation studies on the C.S.F. specimens.

The patient was therefore transferred to the Saint John Tuberculosis Hospital, where intensive antituberculous medication was initiated. It consisted at first of INH 100 mg. by mouth four times daily, PAS 3 g. by mouth four times daily, streptomycin-dihydrostreptomycin mixture 1 g. intramuscularly twice daily (soon afterward reduced to 0.5 g. twice daily), and cortisone 100 mg. intramuscularly once daily. This in essence was the medication used for many months thereafter, with minor alterations from time to time; the PAS was the buffered (sodium-free) type; the cortisone was replaced by prednisone after a time. At no time was there any intrathecal therapy, though follow-up lumbar punctures were carried out at intervals.

On this regimen the patient showed rapid improvement of her cerebral status, and by early January 1958 she seemed to have recovered completely, at least to gross observation, though she was still confined to bed. However, neurosurgical consultation was again requested towards the end of May 1958, soon after the patient had begun to report increasing numbness and tingling in the lower limbs, as well as exhibiting increasingly frequent urinary incontinence, particularly at night. Examination at that time showed the patient to be in excellent general physical condition, conscious, normally oriented and mentally bright. The fundi were normal, except for slight relative pallor of the temporal half of the left optic disc. The pupils were equal, though the pupillary responses to light were moderately sluggish. The upper limbs showed brisk deep tendon reflexes, but otherwise were neurologically negative. However, in the lower limbs there was a moderate to marked degree of spastic paresis, most marked distally; the knee and ankle jerks were hyperactive,

*Presented at Atlantic Regional Meeting, Royal College of Physicians and Surgeons of Canada, held in Halifax, N.S., on October 31, 1959.

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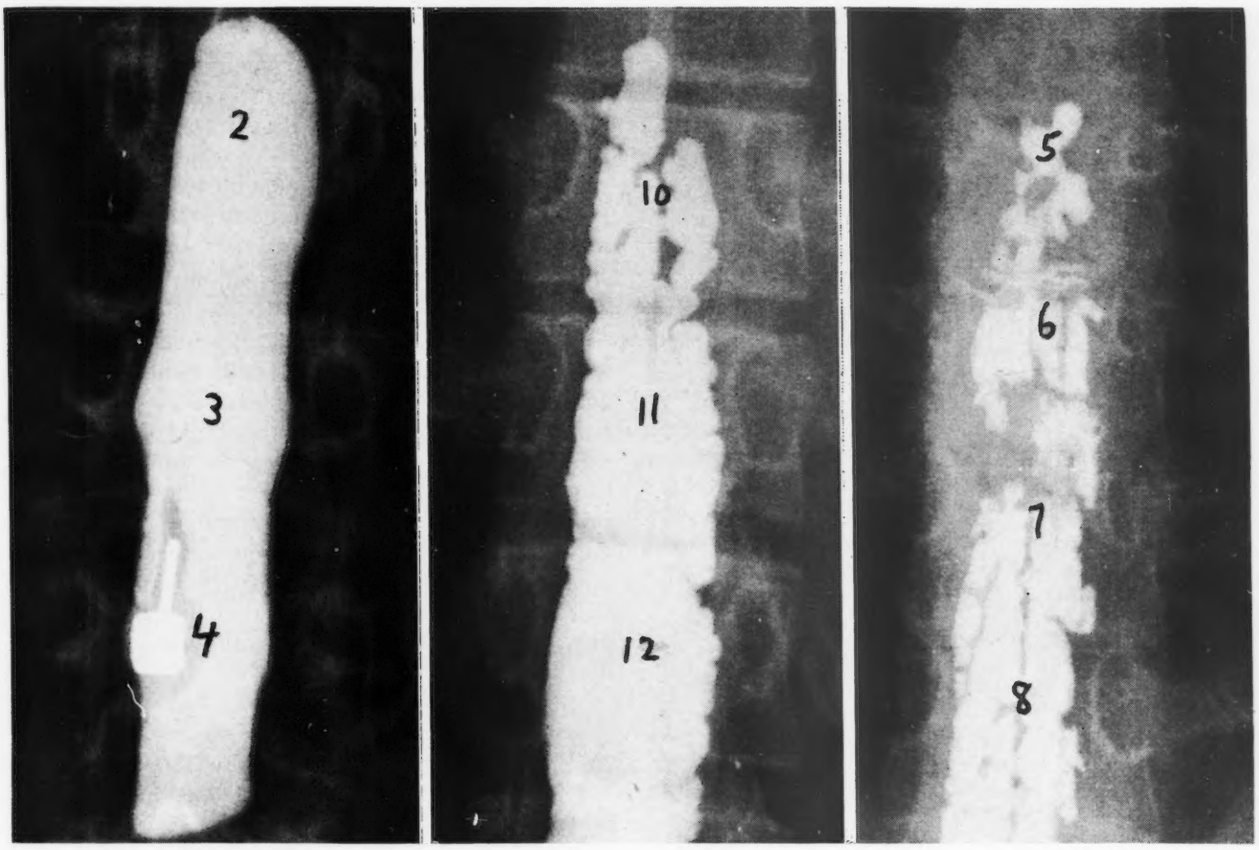

Fig. 1.—Preoperative myelogram, representative spot films. (a) Oil column is normal in lumbar region. (b) Oil begins to break up at T10 level. (c) Oil becomes markedly irregular in mid-thoracic region, and does not pass above T5 level, in spite of extreme head-down posturing.

there was bilateral ankle clonus, and the plantar responses were extensor. Pain sensation was retained throughout, but the patient claimed that its character was a "little different" on the left side, from about the nipple level down, and including the sacral area; position sense was impaired in the toes of both feet. This picture certainly suggested a partial transverse myelitis, involving the upper thoracic spinal cord. However, review of the serial lumbar punctures that had been performed on the patient indicated that there was some suggestion in the more recent punctures of at least a partial degree of intraspinal obstruction.

It was therefore decided to bring the patient back to the Saint John General Hospital for myelography. This examination revealed an extensive irregularity of the oil column from the level of T10 to T5, and none of the oil would pass proximal to the T5 level. This picture was regarded as compatible with an adhesive arachnoiditis and complete subarachnoid obstruction at T5 (Fig. 1). After a good deal of deliberation and some consultation, as mentioned previously, it was decided to explore the upper thoracic spinal canal, and this was done by laminectomy from T3 to T6 inclusive on July 3, 1958.

At operation, the exposed portion of the extradural space did not contain the usually observed amount and type of fatty tissue; this was particularly true at the T4-5 level, corresponding to the level of complete obstruction demonstrated in the myelogram. At no time was any normal dural pulsation observed. When the dura was opened, the arachnoid was found to be markedly thickened and firmly adherent to the underlying cord, forming an inseparable coating over the latter. This thickened arachnoid was, however, only lightly adherent to the dura, so that it was possible to separate the two by careful blunt dissection. At the upper end of the exposure some flow of C.S.F. was observed after the dura had been opened. A small specimen of the abnormal arachnoid tissue was excised for microscopic study, but no attempt was made to separate this thick layer of tissue from the cord, for fear of doing more harm than good, particularly in view of the only partial nature of the patient's preoperative spinal cord deficit. However, the dura was left open, as a possible additional decompressive measure. Microscopic examination of the arachnoid biopsy, as well as of the abnormal extradural tissue obtained from this area, was interpreted as showing a chronic granulomatous inflammatory reaction, with a tendency to "tubercle" formation, though special staining failed to reveal any tuberculous organisms (Fig. 2).

The postoperative course was entirely satisfactory. Immediately after operation the patient claimed that her left leg felt "more like [her] own", and that she was "more normally conscious" of her voiding than before. Indeed, she was never incontinent after the first postoperative day, though some urgency of micturition persisted, lessening gradually in degree. Examination on July 18 revealed definite objective improvement of the preoperative neurological deficit—weak abdominal responses were obtainable for the first time; there was no longer any ankle clonus; the plantar responses were "equivocal" rather than definitely extensor; sensation was (subjectively and) objectively normal in all parts of the body; and motor power and co-ordination were also much improved, particularly

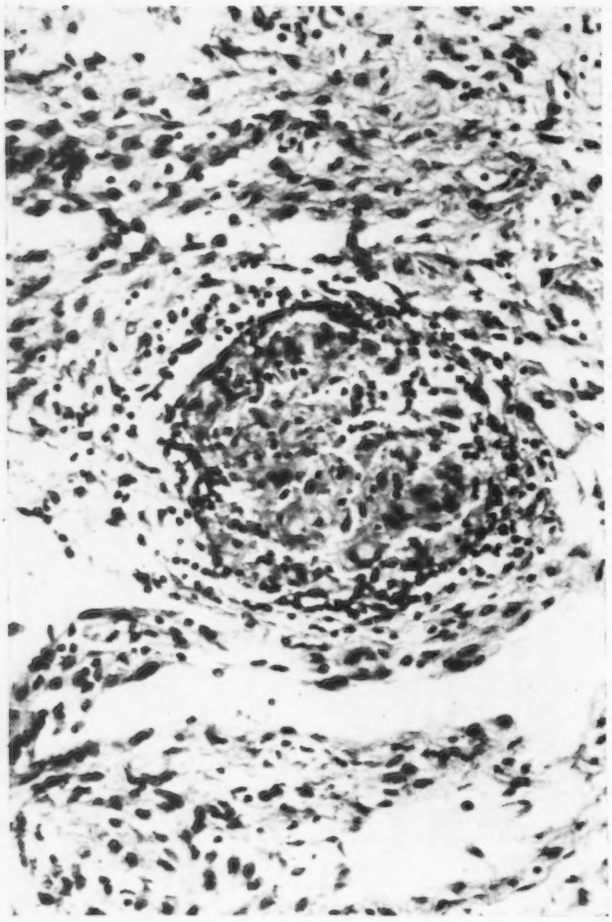

Fig. 2.—Microscopic appearance of arachnoid biopsy, showing chronic granulomatous inflammatory reaction, with tendency to "tubercle" formation. × 64.

in the previously paralyzed feet. Physiotherapy was given regularly. On July 29 (26 days after operation), the patient was regarded as ready for weight-bearing, and she began walking with the help of crutches; these she subsequently discarded, as her balance and walking gait steadily improved.

In September 1959, our patient returned to school, resuming the eleventh grade, where she had been interrupted by the original illness in November 1957. She remained on INH medication, 300 mg. daily, along with pyridoxine 25 mg. daily, until December 1959, when all medication was stopped, two years of continuous antituberculous medication having then been completed. Recent (March 12, 1960) review of the patient revealed only minimal residual neurological deficit. The optic discs showed slight temporal pallor. In the lower limbs there were hyperactive reflexes and equivocal plantar responses, but muscle power and sensation were good. The walking gait was grossly normal, though the Romberg test was still positive and tandem walking was only fair. Questioning also revealed persistence of some urgency of micturition, though bowel function was grossly normal. In general, the patient had no serious complaints, and stated that she was able to pursue her daily activities without significant difficulty. Indeed, she had even returned to her previous scholastic standing near the top of her class.

CONCLUSION

The postoperative improvement of the spinal cord lesion in this case forces one to conclude that such a lesion in a patient with arrested tuberculous meningitis is at least not always due to an intrinsic myelitis alone. That is, in such a case, even without prolonged (or any) intrathecal medication, one may be dealing with a significant obstructive and compressive process in the spinal canal, which may be improved by relatively simple surgical intervention. . . . .

It is hoped that this report will stimulate further active interest in this problem. The information gained from additional case reports such as this should result, as mentioned in the introduction to this presentation, in a clearer delineation of the proper management of those patients with tuberculous meningitis in whom the disease has been adequately arrested by medical therapy, but who still suffer from a neurological deficit sufficient to prevent their satisfactory rehabilitation to a useful life.

SUMMARY

This is the case report of a 15-year-old girl with tuberculous meningitis. The patient's cerebral status improved satisfactorily on intensive specific chemotherapy (without intrathecal therapy). However, at some time during the early months of such medical treatment there appeared evidence of apparently progressive spinal cord dysfunction. Myelography demonstrated an obstructive arachnoiditis in the thoracic region. This diagnosis was confirmed at operation, and was treated by decompressive laminectomy with opening of the dura. Operation was followed by satisfactory though not complete improvement of the spinal cord deficit. Details and illustrations of the preoperative findings, of the gross and microscopic findings at operation, and of the postoperative course, have been presented. The postoperative follow-up is now approaching a full two years.

Survival of patients with adequately treated tuberculous meningitis has become increasingly common in recent years. However, the management of some of the residual neurological deficits in such patients has not yet been clearly delineated, since this is a problem of relatively recent vintage, and with relatively little clinical material upon which a decision regarding such management may be based. Hence the possible value of this presentation.

The help of Dr. I. V. Allen, who supervised the medical management of this patient at the Saint John Tuberculosis Hospital, is gratefully acknowledged.

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SPECIAL ARTICLE

WHY DO PEOPLE SUE THEIR DOCTORS?*

ALAN A. KLASS, B.A., M.D., F.R.C.S.(Edin.), F.R.C.S.[C.], *Winnipeg, Man.*

PINOCCHIO HAS been hanged by assassins, cut down by a falcon and taken to the home of Fairy. The most famous doctors in all the realm have been summoned in consultation. One after another they arrive and place themselves around the bed of Pinocchio: a Crow, an Owl and a talking Cricket. Fairy gives them a brief history of the case and then asks them a simple question: "Is the puppet dead or alive?"

Crow advances first, feels the puppet's pulse, then his nose, then the toe of his foot. Having done this carefully, he steps back and solemnly renders the following opinion: "To my belief the puppet is already quite dead; but if unfortunately he should not be dead, then it would be a sign that he is still alive."

Next the Owl comes forward, examines the patient, steps backward and says: "I regret to be obliged to contradict the Crow, my illustrious friend and colleague, but in my opinion the puppet is still alive; but if unfortunately he should not be alive, then it would be a sign he is dead indeed."

All the while the Cricket stands quietly by. He is a little fellow and except for a tall black hat, balding head and horn-rim spectacles, is quite ordinary in appearance. However, if one looks closely, it is evident he is unimpressed by the performance of his fellow consultants.

"And you – have you anything to say?" asked Fairy.

"In my opinion," replied the Cricket, "the wisest thing a prudent doctor can do, when he doesn't know what he is talking about, is to be silent." And with that he turned and walked away.

When he wrote this story 50 years ago, Senor Lorenzini, by a kind of extrasensory perception, must have been thinking of a doctor addressing himself to a medico-socio-legal problem.

The past 25 years has been a period in medical history made significant by new discovery and higher standards of treatment. Not only the pages of weekly public magazines but even the medical journals are full of the stories of real progress. It seems then a curious paradox that during the same period the number of actions alleging professional negligence against the doctor have risen in most places – precipitously in some. There is little doubt that we are practising better medicine than our fathers and certainly better than our grandfathers. Judging by the increase in actions for negligence, our grandfathers were a careful group not given to error, while we today are a blundering lot of delinquents, and getting worse year by year. Surely this is not the case. Then how account for this increase?

Mainly in the United States and to a lesser degree in the United Kingdom and Canada, the statistics show that more and more doctors have to face the allegation of negligence.

The American situation is chaotic and from the doctors' point of view deplorable. Accurate figures are impossible to obtain. Approximately 6000 claims were initiated in the United States in the year 1958.[1] Compare this with an estimated 103 cases in Canada in 1956.[2] Correcting for difference in population, this gives six times as many cases in the United States. According to the American Medical Association one out of every seven doctors in the United States has been sued. In New York the ratio is one out of five. In California it is one out of four.[3] In the United States malpractice insurance rates have gone up from $40 to $50 annually 20 years ago to $400 to $600 currently, and they are rising annually to compensate for the loss experience of insurance companies.

Here are a few examples from the American scene, glaring no doubt, but they did reach the level of actions instead of being strangled at birth. (a) A suit for a penicillin reaction after standard treatment for gonorrhœa – for two million dollars. (b) In Brooklyn the claim was made that a nine-year-old boy was affected by "strange sex urges" after being treated with male sex hormones. The claim was for a million dollars. (c) Awards in judgments have run in a range up to $115,000, $128,000, $210,000 and $250,000. The record out-of-court settlement awarded in a California action is $290,000.

The situation in the U.K. is not as serious but is rapidly rising to levels that are disturbing many British doctors. Again, figures are difficult to obtain but the statistics from the Legal Aid and Advice Act[4] are informative. Under this act people with a claim for legal action and with an income of less than £750 a year can obtain public assistance toward court action. In 1956, 600 certificates enabling action to be taken were granted in the medical area. The figure does not include actions brought by those who do not qualify under the terms of the Legal Aid and Advice Act and who pursued their actions without state aid. In England losses were £23,000 in 1950 and £159,000 in 1954, a sevenfold increase in four years.[5]

In Canada the situation is not clearly as serious. The Secretary of the Canadian Medical Protective Association stated that 103 threats of legal action[2] were initiated in 1956. At that time there were 16,000 doctors in Canada and 9000 belonged to the Canadian Medical Protective Association, so that there were 103 threats against 9000 doctors. No figures are available for the remaining 7000 doctors.

It becomes understandable, though perhaps unreasonable, that American medical leaders in their alarm have developed a degree of resentment toward their professional brothers – who sometimes become their adversaries – the lawyers. "These malpractice lawyers and their suits – many of which have absolutely no basis in fact – have caused great damage to the medical profession and to the public," said Dr. Paul R. Hawley, Director of the American College of Surgeons. In England,

*Read before the Medico-Legal Society of Manitoba on January 26, 1960.

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too, there is evidence of smouldering resentment. On March 12, 1959, the Medico-Legal Society of London met and debated the question "That the Legal Profession Be Abolished". The Medico-Legal Journal of London also reports a debate with the title "Has the Legal Aid Act Enabled the Lawyers to Feather Their Nests with the Doctors' Plumage?"

These are obviously febrile attitudes reminiscent of the witch hunt. It is not lawyers who sue doctors; it is people. Then why do people sue doctors? What may be some of the reasons to account for this state of affairs, deplorable to the medical profession at large and calamitous to the individual doctor?

First and foremost is the very nature of medical practice itself. In a field of rapid discovery, the accepted treatment of today was risky yesterday; and will be obsolete tomorrow. During the period of yesterday, the treatment was new and hazardous, perhaps experimental in the eyes of the court, and few doctors would come to its defence. During the period of tomorrow it is obsolete, antiquated, proven to be wrong, condemned in the witness box and accepted as negligent by the court. It is only during the brief period of today that the treatment is right and proper, and this period is made even shorter by the rate of discovery.

Take the apparently simple business of staying in bed after a surgical operation. When I was an intern 25 years ago it was the custom and practice to keep a patient immobile on his back for two or three weeks after even the simplest operation. A classmate of mine was seized with the crazy idea that his patient should get out of bed 24 hours after a gallbladder operation. The patient did as ordered, his wound broke open and he eviscerated. He had to have a second operation to resuture his wound. An action was started against this wild young surgeon and he quickly left town and moved to Texas where, instead of practising, he adopted the safer life of marriage to a multi-millionaire oil widow. He could not get a single doctor to support him in his wild scheme of getting patients out of bed so soon after the surgical operation.

That was yesterday. Today any surgeon who does *not* get his patient out of bed on the second or third day after the operation must have a very good reason for it. If by mishap a pulmonary embolism should occur in the patient who is kept in bed for a week or more, the surgeon would be hard put to find support among his colleagues should an action arise. He too may have to take off for Texas and look for his rich oil widow!

Writing in the *British Medical Journal* on "Pitfalls in Medicine",[6] Professor Douthwaite deals with another aspect in the practice of medicine that leads to error and possible legal action. It is in the very nature of disease that it affects no two individuals alike. Even where the diagnosis appears identical, the response to accepted treatment in two individuals may vary from the rewarding to the disastrous. Here lies a fertile area for action. The same surgeon performs the same operation on two individuals. One does well; the other does poorly for no known reason and the patient or relatives or both are unhappy. It is in this unavoidably large group of unhappy patients that actions often arise, even where negligence can not be proven. They arise in this group because of a more or less understandable desire to seek redress and, although they may not reach the courts, they do immediately achieve the status of nuisance value. This nuisance value has become a profitable commodity in California and New York where everyone—lawyers, litigants and insurance companies—seemingly profit at the expense of the doctor. This point will be dealt with later.

Besides the element of discovery and its effect on current practice, and in addition to differing reactions of individuals to similar standard treatment, there is a third factor with which the doctor must contend—the element of the genuine mistake.

> "Mistakes are inherent in the pursuit of a progressive science and not to admit them spells death to any hope of advancement in learning. To be punished for them is equally disastrous to advancement. The layman has difficulty in understanding that the specialist and consultant can make a mistake; in fact he goes further and is only too apt to attack him and his general practitioner in the courts of law, not because he is necessarily aggressive but because he is ignorant in that he will compare the machine which can be torn to pieces with the infinite and variable complexity of the human body."[6]

I am purposely avoiding those straightforward and unfortunate occasions where an overt act has occurred: the sponge or forceps left in the abdomen; the healthy not the diseased kidney removed; the patient being circumcised when it is his tonsils that should have been attacked. In these cases, where I believe the legal principle of "res ipsa loquitur" applies, there is general agreement that the victim should have redress. These are not cases where matters of professional judgment or opinion are seriously at stake.

It is with the more subtle areas of so-called negligence that there is a growing uneasiness among doctors. The word itself to the lay person has a connotation beyond the strictly legal definition. The Rt. Hon. Mr. Justice McNair,[7] speaking before the Medico-Legal Society of London, had this to say:

> "A finding of negligence in Law does not in any way necessarily involve a finding of moral blame or indifference or recklessness, but ordinarily consists in the doing of some act which a reasonable or prudent person would not do or omitting to do some act such a person would do . . . It has been picturesquely said in such cases, the reasonable man is the man on the Clapham omnibus—he has not the courage of Achilles, the wisdom of Ulysses, or the strength of Hercules nor the prophetic vision of a clairvoyant."

It is reassuring that this is true in the language and meaning of the court. But to the doctor "negligence" means something else again. Dr. Alistair French,[4] Secretary of the Medical Protection Society of England, had to say that in his job he was aware of a very great indignation among many doctors to the attitude of the lawyer towards this question of professional negligence. A doctor feels that he like anybody else could make a

mistake and he was indignant that he should be especially penalized for so doing and be accused of negligence and get all the unpleasant publicity which the trial of such an action entailed. It was extremely difficult to convince a lay person, be he judge or juryman, that a doctor was ever permitted to be in the slightest degree careless. In these days with the tremendous advances in the art and science of medicine, the public tended to expect a doctor to guarantee a cure or a good result in every case, and it was very difficult to convince that that was not so and that, however conscientious and skilled a doctor might be, such a result could not be guaranteed.

It is in this realm of actions resulting from genuine mistakes or errors in judgment (rather than the forceps in the belly) that doctors show such resentment to the word negligence, a word, according to Prof. Ian Aird, that has an "ugly flavour". The diagnosis of cancer wrongly made or wrongly not made; injuries that in the judgment of the attending practitioner require no x-ray and later turn out to have included a fracture, are examples. It is perhaps unnecessary here to plumb the levels reached by American courts[8] where an obstetrician was deemed negligent because he found it impossible to be present at both of the simultaneous confinements of two of his patients. It was ruled that he used faulty judgment in assuming care of both patients some months previously!

Why do people sue doctors? Having considered some of the factors as seen through the eyes of a doctor, there is still the basic question: why? why? After all, grievance, negligence, malpractice are not new things in the relationship between patient and doctor, and yet we have this monster of litigation growing at a fantastic rate in the U.S.A., more slowly but none the less growing in the U.K., and perhaps stirring towards growth in Canada.

There are special reasons in the U.S.A. The primary one is the jury system of trial to which every American is by constitution entitled. "There is an alarming tendency," says R. Crawford,[3] a Cleveland expert on malpractice law, "for the courts to rule for the patient who is apparently injured, not because of any negligence on the part of the doctor, but because they feel sorry for the patient." Couple this sorrow with what may charitably be called a certain difference in ethical standards of some members of the legal profession in the U.S.A. and you have a dangerous combination.

In England perhaps the weakening of traditional patient-doctor relationship produced by the National Health Service and abetted by the operation of the Legal Aid and Advice Act (in which lawyers are paid 85% of their fee by the state) may in part account for the increase.

None of these reasons by themselves would be important were not suing doctors—on the whole—a reasonably safe and profitable adventure. The universal principle that you can not "draw blood from a stone" works here in the reverse. Given the combination of an individual and a lawyer both prone (for whatever reasons) to take action against a doctor, there is only one further question: has the defendant enough money to satisfy a judgment? And here is where the new phenomenon, growing fantastically in the past 20 years, comes in—the Insurance Company.

Along with the entirely reasonable motive of profit, the insurance company has the best intent in the world—to insure the doctor against financial calamity. And yet, paradoxically, by the very provision of this financial bulwark against catastrophe, an answer is automatically provided to that sixty-four dollar question: "Is there enough money to satisfy judgment?" Of course there is, and one sues not the kindly, perhaps bumbling doctor, but a slick and dehumanized corporation of steel and concrete with millions. In the opinion of many, this is the prime factor that has accounted for the fantastic growth of actions against doctors in the U.S.A. and the increase in the United Kingdom. This is the factor that may well raise the same monster in Canada.

The actors in these plays, multiplied by the hundreds, are as follows: a plaintiff with a grievance that may vary from the ridiculous to the very real injury of the "res ipsa loquitur" variety, a lawyer who knows his job, a doctor eager to settle to avoid the embarrassment and injury to reputation of any court action no matter how right he may be, and lastly an insurance company reluctantly willing to settle. Reluctant because no company likes to take a loss. Willing because ultimately next year's premiums (in other words, next year's business) will be determined by the profit and loss statement of this year's operation. Premiums can go up and do go up and while the loss may appear formidable on the books, the "cash-flow" increases year by year. These are cost-accounting subtleties, but it is surprising how insurance companies thrive in spite of reported substantial losses in certain specific fields of operation.

The medical profession in Canada is indeed happy and fortunate that so far this country seems to be an oasis of sanity in this increasing clamorous warfare between litigant and lawyer on the one hand and doctor and insurance company on the other. Will it always be so? To one who practised in England 20 years ago, it seems incredible that such a state of affairs could have arisen. England, where the doctor seemed to be held in very special esteem and where the Bar and the courts in the eyes of many are the best in the world. But it did happen. Will it happen here?

It would be comforting to express optimism, but a realistic look into the future disturbs the lull of a false security. In Canada too there is inevitable and growing government involvement in hospital and health planning. We have not travelled the distance of the United Kingdom, but we have set off in that direction. Because of the recent change in hospitalization, there is already a difference in suing a hospital with the involved doctor. Yesterday the citizen had to consider suing a group of voluntary citizens who made up the corporate board of a hospital that was perennially in desperate need of money. This used to be the target of any projected suit. Today it is the government. As witness England, there is in the mind of the public a distinct difference.

What can be done to prevent this situation arising in Canada? Here the doctor must bow to his brother learned in the Law. It is no accident that

in the Parliament at Ottawa in 1957 there were 154 lawyers in the House. It is the legal profession that makes up the majority of the Law Amendments Committees of the Legislatures. By profiting from examples in other countries, could not some modification be made to existing rules regarding actions for professional negligence that would be fairer to members of the profession without doing violence to basic rights? Only the learned profession of Law can grasp this nettle.

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SHORT COMMUNICATION

ETIOLOGICAL INVESTIGATION OF ARTERIOSCLEROTIC HEART DISEASE

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FIFTY-NINE unselected patients who were seen in the heart clinic of the out-patient department of the Toronto Western Hospital with a diagnosis of chronic arteriosclerotic heart disease were investigated with regard to a family history of arteriosclerotic heart disease, cerebral vascular disease, diabetes mellitus and gout; a past history of diabetes, gall stones, gout and nephritis; past and present weight; the presence of hypertension; and dietary habits with regard to animal fats. If their diet contained less than 90 g. of fat per day, it was considered normal; and over 90 g. of fat per day was considered to be a high fat diet.

TABLE II.—SIX CASES OF ELEVATED BLOOD URIC ACID

No.	*Sex*	*Age*	*Blood uric acid, mg.%*	*Serum cholesterol, mg.%*
1	M	44	4.4	334
2	F	55	5.2	342
3	M	66	4.4	301
4	M	66	5.6	256
5	M	69	6.3	128
6	M	76	5.0	255

RESULTS

The series comprised 33 males and 26 females (Table I). There was a family history of arteriosclerotic heart disease in 21 cases, a family history of cerebral vascular accidents in 12, and of diabetes mellitus in four. In no case was there a family history of gout.

The past history of these patients showed that 17 had diabetes mellitus, and 11 of these were female; seven had gall stones, and these were all female.

Six patients had an elevated blood uric acid (normal 2-4 mg. per 100 ml. of blood). Only one of these had a high blood uric acid; this was a male, aged 69, with a blood uric acid of 6.2 mg. per 100 ml. and a serum cholesterol of 128 mg. per 100 ml. He was attending the arthritic clinic for arthritis in one knee. The other five had no history of gout or arthritis, and there did not appear to be any relation between the blood uric acid and the serum cholesterol values. There were no cases with a past history of nephritis.

Thirty patients were or had been overweight.[2] Hypertension was present in 15, and this was fairly equally divided between the two sexes. There were 29, 18 men and 11 women, who had a diet high in animal fat for most of their life. This finding was noted most in the age group 44-54 years in which there were 16 cases and 11 cases on a high fat diet, and 10 of the 16 were overweight.

The serum cholesterol level was elevated above 250 mg. per 100 ml. in 11 males and 9 females. There were 5 females and 4 males with values over 300 mg. per 100 ml. Xanthelasma palpebrarum was observed in 2 females and the serum cholesterol values were 126 mg. in the one and 378 mg. per 100 ml. in the other.

TABLE I.

			Family history				*Past history*				*Diet*					
Age	*Sex*	*No. of cases*	*Arterio-sclerotic heart disease*	*Cerebro-vasc. disease*	*Diabetes*	*Gout*	*Diabetes*	*Gall stone*	*Gout*	*Nephritis*	*Normal*	*High fat*	*Over weight*	*High blood pressure*	*Elev. blood uric acid*	*Elev. serum cholesterol*
44 to 54	M	12	3	2	0	0	3	0	0	0	4	8	6	1	0	6
	F	4	2	2	2	0	2	0	0	0	1	3	4	1	0	0
55 to 64	M	7	4	2	0	0	0	0	0	0	4	3	2	4	0	3
	F	15	5	2	2	0	5	4	0	0	8	7	9	3	0	7
65 to 76	M	14	3	3	0	0	3	0	1	0	7	7	5	1	6	2
	F	7	4	1	0	0	4	3	0	0	6	1	4	5	0	2
		59	21	12	4	0	17	7	1	0	30	29	30	15	6	20

CONCLUSIONS

There is a genetic factor present in many patients with arteriosclerotic heart disease that was evident in 35% of this group. Diabetes mellitus was found in 27%, which was in keeping with the finding that of 100 consecutive deaths in the diabetic clinic of the out-patient department of the Toronto Western Hospital more than 60% were due to arteriosclerotic heart disease.

Overweight was found to be a factor in 50% of the cases, a high-fat diet in 48%, and hypertension in 25%. In the age group 44-54 years, overweight was noted in 60% and a diet high in animal fat in 67%. One female was known to have had arteriosclerotic heart disease since age 35, at which time she had an acute myocardial infarction: she had normal endocrine function but had lived almost entirely on a diet of fried hamburgers and fried potatoes.

Dock[3] has commented on the high rate of atherogenesis in young men and has stated that this may be due to a high animal fat diet, as suggested by the findings of Young and Pilcher[4] in a dietary survey in upstate New York in 1948.

The serum cholesterol level was elevated in 33% of cases but only markedly elevated in 15%. Hyperuricæmia was not a common finding, occurring in six cases (10%); it was markedly elevated in only one case (1.7%). This is in marked contrast to the report of Kohn and Prozan,[5] who found that 20 out of 35 males and 6 out of 15 females with acute myocardial infarction had a serum uric acid level above 6 mg. per 100 ml. They were reporting on cases of acute myocardial infarction, whereas this report was based on cases of chronic arteriosclerotic heart disease.

SUMMARY

A family history of arteriosclerotic heart disease,[6-8] the association of diabetes mellitus, the clinical finding of overweight[9, 10] and hypertension,[12, 13] the history of a high animal fat diet,[9] and the biochemical finding of hypercholesterolæmia[14] are all common findings in patients with arteriosclerotic heart disease. However, many, some quite young, have this disease without any of the above being present. Hyperuricæmia was a rare finding in this group studied.

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Canad. M. A. J.
Aug. 13, 1960, vol. 83

THE CANADIAN MEDICAL ASSOCIATION
JOURNAL
LE JOURNAL DE
L'ASSOCIATION MÉDICALE CANADIENNE

published weekly by
THE CANADIAN MEDICAL ASSOCIATION
Editor-in-Chief: DONALD C. GRAHAM, M.D., F.R.C.P.[C.]
Managing Editor: T. C. ROUTLEY, M.D., F.R.C.P.[C.]
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(Information regarding contributions and advertising will be found on the second page following the reading material.)

PULMONARY FUNCTION AND THE CONCEPT OF CHRONIC NON-SPECIFIC LUNG DISEASE

THE status of measurements of pulmonary function has undergone interesting changes during the past decade. In the early years of this period, there was a sharp rise in the frequency of surgical treatment for tuberculosis and other destructive pulmonary diseases, especially in the United States, Canada, the United Kingdom and the Scandinavian countries. Many patients with borderline pulmonary function were so treated in an effort to remove from circulation the reservoir of open tuberculosis, from which it was considered by public health authorities that the largest proportion of new cases arose. Our surgical confreres became quite adept at the extirpation of lobes, segments, sub-segments and "wedges" of irreparably destroyed lung tissue, and, in many cases, whole diseased lungs were removed. It soon became clear that some accurate measure of pulmonary function was required in order that the diseased organ might not be too widely resected, leaving the patient with insufficient lung tissue for survival. It was obvious, of course, that the measurement of total pulmonary function (i.e. the function of both lungs together) was unsatisfactory for this purpose, and that the function of individual lungs would have to be determined. This of course led to the introduction of differential bronchospirometry. It must be emphasized here that, in some centres, such elegant facilities were not available, and that, in these centres, phthisiologists and thoracic surgeons were forced to rely on less esoteric procedures such as physical examination, simple ventilatory studies, and fluoroscopy for their final decisions. It still remains to be seen whether the surgical results based on the more complex pulmonary function studies have been happier than those derived from the simpler tests. Be that as it may, certain changes have taken place in our concepts of the management of destructive pulmonary diseases, tuberculosis in particular. The trend now appears to be in the direction of prolonged rest and antimicrobial therapy (with greater or less rest, depending on the individual attitude of the phthisiologist), and a larger proportion of patients now are spared surgery than was the case in the past. For these reasons the measurement of pulmonary function in tuberculosis has fallen into partial disuse, and emphasis has shifted to its employment in non-tuberculous pulmonary disease.

It should be stressed here, however, that, from the standpoint of function, the lung, like the nervous system, can respond in only a limited number of ways, without reference to the disease process that has brought about such response. A recent CIBA Guest Symposium[1] has suggested that the diagnoses "chronic bronchitis", "asthma", and "emphysema" are used without any general agreement on the clinical conditions to which they refer. It seems that any one (or more) of these terms may be used by different clinicians to describe the same condition. It also appears that the term "chronic bronchitis" is often used in the United Kingdom to describe situations that would be classified as "asthma" or "emphysema" in Canada and the United States. The participants in this symposium therefore suggested that the term "chronic non-specific lung disease" be used for this whole group. It is emphasized that "chronic non-specific pulmonary disease" may coexist with any of the well-known destructive pulmonary diseases. For example, in a case of healed pulmonary tuberculosis or of simple pneumoconiosis, the symptoms may be due, not to the original disease but to the *results* of that disease, i.e. to "chronic non-specific lung disease". This designation, therefore, merely represents the recognition that, as already indicated, the lung, like the nervous system, can respond functionally in a limited number of ways to disease. It would seem that this is a rather useful concept, provided one keeps in mind that different forms of chronic non-specific lung disease affect different pulmonary "functions" in different ways. Thus we have ventilatory defects, alveolo-capillary defects or blocks, and circulatory defects, i.e. impairment of function in chronic non-specific lung disease may be the result of abnormalities in ventilation, perfusion and diffusion.

The participants in the symposium also point out that, despite the fact that the abnormalities described fall into a small group of disturbed functions, the tests that must be done to assess the status of the various pulmonary functions are large in number. They include, among others, certain tests of ventilatory functions such as vital capacity; one-second forced expiratory volume (before and after inhalation of a bronchodilator aerosol); the usual subdivisions of lung volume by open- or closed-circuit methods or by body plethysmography; forced inspiratory spirograms for comparison with the expiratory spirograms; measurement of unevenness of ventilation with the nitrogen or helium meter; measurements of the distribution of pulmonary ventilation and per-

fusion; measurements of pulmonary compliance, airway-resistance and work of breathing by œsophageal pressures and other means; measurements of diffusing capacity for oxygen or carbon monoxide; measurements of the ventilatory requirement on exercise; measurements at rest of the arterial blood gases and pH; and assessment of distribution of the pulmonary circulation by clinical, radiological and electrocardiographic studies. Although not stated, it is implied that, in obscure cases, cardiac catheterization will have to be resorted to.

It would seem, therefore, that pulmonary function measurement has expanded tremendously in the past few years and that, in doing so, it may have lost a great deal of its practical application. It may have become, and desirably so, primarily a research tool that will aid us in attaining a broader and clearer understanding of pulmonary physiology. Nevertheless, one asks oneself the question, "What of the quest for objective pulmonary function tests that will measure such function in individuals seeking pensions for war-aggravated 'bronchitis', industry-caused pneumoconiosis, etc.?" It is clear that one of the essential stimuli for the elaboration of more and more complex pulmonary function tests has been the search for such objective data. It would seem that, despite the suggestions of physicians associated with industrial and pension-granting organizations, this desirable state of affairs has not yet been reached. In a paper[2] distinguished by its objectivity and breadth of statistical evaluation, Shepard and Turner have attempted to consider the diagnostic limitations of pulmonary function tests. In a disease syndrome, characterized by interstitial fibrosis and giving a mean functional loss of some 20%, they found that a single test, albeit a rough one, i.e. vital capacity, showed an overlap of 22% between normal and abnormal subjects. Although one admits freely that the vital capacity is considered by most workers in chest disease to be an extremely rough approximation, it would seem, nevertheless, that even a *battery* of tests does not provide the desired answer. These workers, in addition, performed eight other tests on normal and abnormal subjects, including (a) one-second forced expiratory volume, (b) maximum inspiratory and expiratory pressures, (c) carbon monoxide uptake and functional residual volume, (d) peripheral arterial oxygen saturations and (e) the estimation of diaphragmatic movement from full inspiratory and expiratory films and expressed as a ratio of the radiological chest length. They then subjected their data in these investigations involving 14 abnormal and 18 normal subjects to a formal discriminative analysis. They found that this combination of tests reduced their overlap from 22 to 15.2%, but the improvement was not statistically significant. They finally state, "it seems probable that the main factors limiting the discriminatory capacity of laboratory tests are the underlying physiological variation and *redundancy* in added tests."

It would appear, therefore, that three situations must be faced. Firstly, despite the fact that pulmonary function tests have become more delicate, time-consuming, and presumably accurate, they must still be interpreted broadly, and in the light of the clinical situation. Secondly, we are not yet in a position to assess "pulmonary function" in a completely objective manner, particularly in individuals who, for one reason or another, are desirous of masking the actual function of their lungs. And finally, as a corollary of all this, it would appear that we have not yet reached the stage of "punch-card diagnosis", even assuming that this were desirable—a highly debatable suggestion.

S. J. SHANE

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PROBLEMS OF SCIENTIFIC TRANSLATING

A report on scientific and technical translating published in 1957 by the United Nations Educational Scientific and Cultural Organisation (UNESCO) deals exhaustively with the general problem of enabling scientists to exploit knowledge published in languages other than their own. The problem of translating medical publications from one language into another is dealt with, and it is shown in one table that of a total of 193 medical periodicals out of 1000 selected at random, 78 were published in English, 22 in French, 28 in German, 13 in Italian and 13 in Russian and other languages using Cyrillic script. Another table of medical periodicals shows that of a total of 4454 publications, 2061 are published in English, some 600 in Spanish and French, somewhat fewer in German, and the number progressively decreases to 85 in Russian.

It is true that publications in many countries, including Russia, abstract their important articles in English, and this is of great help. If, however, the scientist wants to read the original article he is faced with the problem of translation. Three methods are available for tackling the problem of translation: (1) the development of electronic translation, (2) the creation or expansion of a class of professional translators, and (3) the provision of dictionaries and other facilities for scientists who wish to read Russian for themselves. Smyth[1] of Sheffield, England, who reviews the latest Russian-English medical dictionary by Jablonski and Levene, has no doubt that electronic translation will provide at least part of the solution. It is interesting to note that although electronic

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translators have been developed only in the last decade, there are some at present available in Russia that translate French into Russian, and in the United States and in England, others that translate Russian into English. An IBM punched-card computor set up in America can translate short passages of Russian with a 250-word vocabulary and six syntactical instructions. An increase in the number of professional translators of Russian into English is also going to provide added help in this direction, but it is not going to be more than a partial solution. In order to provide adequate translation, the translator has to be proficient not only in the language, but also in the subject matter. The final solution, that of the scientist's learning Russian just as he used to learn German a generation ago, requires, first and foremost, good dictionaries.

Smyth believes that the present dictionary by Jablonski and Levene has some important shortcomings, one of which is the limitation of this dictionary to medical terms. This requires an additional non-technical dictionary to help out with non-technical terms. Other available dictionaries combine both technical and non-technical words. Another difficulty facing the translator is the Cyrillic alphabet used in Russian, but this is far from insuperable. The words of Western origin which are found in Russian writing of the earlier part of the century are, more and more, being replaced by words of Slavonic origin, and this development is likely to continue in view of the increasing national consciousness of the Russian scientists. Similar developments have been known to occur in all other nations in the course of their progress. Smyth also points to the difficulty with abbreviations which are being used to a large extent by Russian scientists. There is a real need for a dictionary containing most of these abbreviations, which are well known within Russia but can present great problems to a translator. Smyth concludes his review by saying that the complete, perfect dictionary of Russian-English medical terms is yet to be published.

A difficulty which has not been mentioned in either the UNESCO report or the review by Professor Smyth is that of semantics. The "iron curtain" which has in reality existed for at least forty years, and has to a large extent existed for centuries, has led to the development of concepts in Russia that are not generally understood outside its borders. In medicine one can think in particular of the concept of "nervism" which is an outgrowth of late Pavlovian thinking and to which many other ideas, not necessarily closely related, are attributed. While these may be completely understood by a Russian scientist, they are found difficult to grasp by those unfamiliar with Soviet terminology. In the socio-medical field too, other problems become apparent as one peruses writings regarding the organization of health services in districts of Soviet Russia. Certain occupations related to medicine such as that of "feldscher"—a male nurse with responsibilities far greater than those of a district nurse and with training which is well below that of a recently graduated doctor—are examples of this. The translator has to be well informed about cultural trends as well if he is to understand fully the meaning of some of the Russian medical writings.

The best answer to understanding Russian medical writing lies in frequent and free personal exchange of information and ideas between scientists on both sides of the "iron curtain".

W. GROBIN

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ASSISTANT TO THE EDITOR

The observant reader will undoubtedly have noted that, with the current issue of the Journal, our masthead has undergone some face-lifting. While never doubting for a moment that the *Canadian Medical Association Journal* has any reader who is not observant, the thought occurs that there may be those too busy and harassed by the toil and tribulations of the day to have registered this minor metamorphosis on their retinæ. In any event it is our pleasure to draw attention to the addition of the name of Robert Randall to those occupying the hallowed space above this column.

Mr. Randall has been a vital cog in the editorial machinery of this periodical for over five years. Unlike many who demand immediate recognition before they have proved their worth, he has laboured long and faithfully, ploughing through prodigious volumes of work week in and week out, without complaint. His skilful hand is responsible in no small part for the final product which we present to our readers.

To the uninitiated, the minutiæ of procedures involved in transforming hieroglyphic-littered manuscripts, editorials, news items, abstracts, book reviews, photographs, charts, obituaries and the hundred and one other viscera of a journal such as this, into a format which lends itself equally to careful reading or casual scanning, are matters beyond comprehension.

In his quiet and unobtrusive way, without fuss or fanfare, Mr. Randall works his weekly miracle with efficiency, imparting his own particular touch here and there to improve the literary quality of the editorial material.

Bob Randall is indeed an Assistant to the Editor and we are pleased to accord him recognition as such.

ED.

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LETTERS TO THE EDITOR

MEDICINE, GOVERNMENT, AND THE FUTURE

To the Editor:

It was my privilege to sit with the General Council of the Canadian Medical Association for two days at Banff, and I heard all the discussion about the threat (?) of state medicine in Canada, notably in Saskatchewan, which has had a bad time, lately. There seemed to me more than once to be a note of serious anxiety, almost of fear, about the future of medicine in Canada, as if we face grave dangers of one kind or another.

I have been associated with health insurance in one way or another since 1924, when I was sent to Ottawa to present the views of the B.C. profession before a meeting of medical health officers from all parts of Canada, and what I said appears in the *Canadian Medical Association Journal*, 15: 287, 1925. It was very immature, in the light of our later experience.

Then, in 1934-35 I was a member of the committee under Dr. Thomas McPherson's chairmanship, appointed by the Council of the B.C. College of Physicians and Surgeons: this committee met members of the B.C. Cabinet (Dr. Weir being Chairman of their Committee). We held innumerable meetings with them, with little or no resulting agreement, and learned a good deal. We had experts to advise us, and so did they: the cost to us amounted to some $15,000, of which part was contributed by the Canadian Medical Association. We were in complete touch with the general profession, were strongly united, and spoke with one voice. In this committee, too, we had strong leadership from Dr. Wallace Wilson of Vancouver, later President of the Canadian Medical Association, to whom was assigned by Dr. McPherson the duty of presenting the general views and final decision of the profession of B.C.

Through the years I have, naturally, come to certain conclusions about health insurance, government-controlled medical plans, state medicine, and so on, and I should like to put these down, for what they are worth.

There seems to me, first, no reason for panic, or for any fear. There is room though for calmness and patience and even generosity.

There are certain fallacies which I should like to point out; some of them came up during the discussions at Banff.

Firstly, the fallacy that *we* ourselves must have a plan to present to the people or their representatives, when they demand complete coverage.

I submit that we have a plan already, which we ourselves put into being—it works; it is fair; it is growing; it is based on the best patterns of practice; it makes for good medical care. Certain elements of the community cannot afford it, but if the government wants to be helpful, or to spread the coverage to everybody, let it provide funds to help this section of the community. However, it must not get these funds by picking our pockets. Sixty to seventy per cent of the community can afford our plan; let them pay for it, and know that they are paying, and how much they are paying. That is good in every way; it lessens abuse of the scheme.

In the course of my paper given in 1925, and endorsed by the B.C. profession at that time, I said, "We in British Columbia do not think that the medical profession should strive to inaugurate any scheme of health insurance, but that we should educate ourselves thoroughly with regard to all its advantages and disadvantages and remain receptive, but ready to meet any situation that may arise."

I have seen no reason for us to change from this position. It is not for us to make any plans. It is not for us, as a profession, to protect the health of the people, and every time that we try to do this, as in our battles with cults and untrained men of various sorts, we get nowhere, and we lose dignity and standing.

The protection of the public is the business of the government, which on many occasions has forgotten this fact. Mr. Douglas is one of the forgetters in this regard, when he threatens us with abolition of the Medical Act, or its alteration as to control.

If and when the people want (as they have a perfect right to want) a wider medical coverage, including everyone, let them, through their representatives, work out a plan and suggest it to us, as the B.C. Government did in 1934-35.

When we have heard and seen the plan, let us then, using as a guide our principles, and the beliefs on which these are based, examine it, and say whether or not we can practise medicine under this plan, as we did in 1934-35. We found it impossible to accept the B.C. Government's plan, and said so, but this does not mean that we are necessarily opposed to *any* plan.

The medical profession in Canada has discussed this fully. We now have a definite set of beliefs and principles in writing, and there is, we firmly believe, practically 100% unity among practising physicians.

Other fallacies are:

1. That there are only two alternatives. The *status quo*, i.e. the free practice of medicine, and state medicine. This last phrase (state medicine) was heard so often at Banff. This is, of course, not true.

We could continue as we are, extending prepaid plans, with subsidization of the indigent, the low wage earner, etc., by the Government. We could have a plan under the control of a Commission, like the W.C.B. We could have a scheme such as they have in Australia or Sweden. These work, apparently, to general satisfaction, and they seem fair to the doctor. They contain checks against abuse either by the doctor or by the patient. They are operated with a minimum of government control.

Lastly, we could have Mr. Douglas' scheme, or something like it, as far as he has suggested a scheme, and work under the Minister of Health.

But it is *for us* to accept or reject *any* scheme, when we have heard it.

2. Another fallacy is that we must be always ready to *fight* anything the Government suggests. There is no need of this; it is *our* government too. We can be willing and ready to meet the Government always, and discuss everything with them, politely, courteously, and in friendly fashion. We did this in B.C. and there was a most friendly feeling, though we had to disagree in the end.

3. Another fallacy is that we have all the rights on our side. We have not. There is a situation which requires a remedy; that illness and its costs, and loss of time, are an intolerable burden to many, and they

need help. This is greatly exaggerated, of course, by the proponents of compulsory health insurance, state medicine, etc., but it does exist, and the Government, speaking for the people, has a right to alleviate this situation.

On our part we must be willing to listen to any suggestions made by the Government; we must weigh these calmly and judiciously in the light of our principles laid down at Banff. (As a matter of fact they were laid down some years ago, but needed simplifying and restatement.)

We must be receptive, even generous.

We must keep out of politics.

We must work as a united body.

We must keep our heads and our tempers and avoid publicity and public relations organizations as much as we possibly can. Let the other side do the talking—while we listen and decide what we can or cannot do.

4. The last fallacy is that the Government is all-powerful, and we *must* accept what they give us, or suffer disaster.

This is still, we often say, a free country, and the medical profession has as much right to maintain and present its views on a united front, as any business or any labour union. We have no Stalins or Hitlers, and we cannot be forced, as a profession, to accept any scheme. But, having said this, we must recognize that progress means change, always; that the rights of all transcend the rights of the few, or rather must be considered equally, and that it lies with us, as it has always lain with us, to give our highest skill and our best services and our work to all who need it, without limitation by poverty, creed, race, or social status, and with generosity. We are able, and should be willing, to help the Government to find an answer to these social problems, and surely this can be done with justice and fairness to all.

J. H. MACDERMOT, M.D.

Vancouver, B.C.

IN THE INTERESTS OF THE COLOUR-BLIND

To the Editor:

In all, for over eight years we routinely used Ishihara or the Dvorine pseudo-isochromatic plates as a part of routine examinations of children, some in Grade IV, but most in Grade VII. At first the children, as a group, filled in a form similar to that described by Heathcote *et al.* (*Canad. M. A. J.*, 82: 1262, 1960) as they read the chart placed on the blackboard. Later the children were tested individually as they were examined.

It happened that the writer's hobby has been colour photography for the past quarter-century. In 1939 we attempted what Heathcote *et al.* successfully achieved. At that time we failed in that certain children could read our projections perfectly, but not the charts directly. Further, replacement of a burned-out bulb in the projector altered the proportions who could not read the projected slides.

The colour testing was discontinued six years ago because:

1. There was no remedial treatment and in many instances, particularly among children who had thus far successfully concealed from others their defect, there appeared to be a bad psychological effect.

2. There were children definitely colour blind, at least to red-green, who did very well with colours. For instance, one child who was markedly colour-blind constantly won prizes in pastel and water colours. Almost invariably the colour-blind, for instance, did well with traffic signals.

3. We found, when fluorescent lighting was introduced, many of the colour-blind and their parents—for we attempted to test the parents of the colour-blind when these were discovered—if colour-blind, would be colour-blind by daylight tests or by incandescent light, but correctly answered the colour charts if the lighting used was fluorescent. We strongly feel that this aspect of colour blindness should be investigated further because it may be that the efficiency of traffic lights could be greatly improved by the use of a fluorescent light behind the colour screen. We have often wondered how frequently those charged with passing a red light are colour-blind. Certainly tests of those so convicted are in order as part of the legal procedure. Society has no right to lay an impossible burden upon them in daily living.

Further, in industry, colour testing should be done under conditions of lighting actually used at work.

One further comment: We routinely now use an audiometer to test hearing. Occasionally we encounter children who are not lip readers, who hear the spoken voice of the examiner, parents, teachers and others apparently normally, who are usually above average intelligence by test and doing well in school, yet who repeatedly, on three or more tests, show a hearing loss of more than 50 db. They are similar, as far as test result is concerned, to colour-blind children who are doing well with their colour problems, for example the colour-blind child artist.

May I again emphasize this plea for the so-called colour-blind. To reject from employment a highly trained boy who may see colours efficiently enough under a different type of lighting, as he apparently did in order to pass his course, is wrong. The history of medicine is filled with overemphasis on tests that, applied too strictly, have unnecessarily altered the lives of the victims, the thousands rejected during the war solely because of an x-ray film who later proved healthy, and vice versa, the difficulties encountered daily in ECG interpretation, etc. Colour tests must be sensibly applied and interpreted. Certainly, where the lives of others must depend upon correct colour interpretation, individuals who cannot pass colour tests should be rejected, but I am sure there are many individuals, colour blind by test, who have learned to efficiently handle many jobs in which the interpretation of colour is required.

GRIFFITH BINNING, M.D.

Saskatoon, Saskatchewan.

CHANGE OF ADDRESS

Subscribers should notify the Canadian Medical Association of their change of address one month before the date on which it becomes effective, in order that they may receive the Journal without interruption. The coupon on page 39 is for your convenience.

Medical News in brief

ETIOLOGY OF BRONCHOLITHIASIS

By the use of known reliable culture techniques and by special stains of histological material, Weed and Andersen examined 12 broncholiths obtained from nine patients (*Dis. Chest*, 37: 270, 1960). By means of the silver chromate technique, they were able to demonstrate bodies that had the morphological features of *Histoplasma capsulatum* in five stones from five patients, and by means of Gram's stain and other stains they were able to demonstrate branching filaments whose morphological appearance was compatible with that of *Nocardia asteroides* in eight stones from five patients. In two stones (two patients), structures resembling both Histoplasma and Nocardia were present in generous numbers. Staphylococci were found in large numbers by culture in two stones; by means of Gram's stain, they were shown to be only on the surface and were presumed to be contaminants and not the original stimulus to the deposition of calcium.

It would thus appear that there is a close relationship between pulmonary mycoses and broncholithiasis.

JEJUNAL BIOPSY IN ADULT CŒLIAC DISEASE AND ALLIED DISORDERS

Jejunal biopsy was carried out in 58 patients attending the steatorrhœa clinic of a general hospital. For purposes of comparison and for determining the normal structure of the jejunal mucosa, biopsies were also performed on 65 subjects of comparable age groups, such as normal medical students, and patients with pernicious anæmia, iron deficiency anæmia, steatorrhœa due to pancreatic disease, regional enteritis and enterocolitis, and miscellaneous disorders not associated with gastro-intestinal disease. In the group with "idiopathic steatorrhœa" Fone *et al.* (*Lancet*, 1: 933, 1960) report that 27 patients had a mucosa whose surface was almost completely flattened and no normal villi were present. The same picture was seen in patients both during a clinical remission and in the initial illness or relapse. In this group 11 patients were treated by a gluten-free diet and of these three were unable to adhere to it. Eight patients had satisfactory clinical remissions. Of the remaining 16 patients, all had regained good health after administration of oral folic acid or parenteral iron, except for one who died of a pulmonary infection.

Twenty-seven other cases also showed an abnormal microscopic appearance; villi were present and their surface was not flattened. In some there was much disturbance in the architecture of the villi and moderate or heavy cellular infiltration, but in others the main feature was shortening and broadening of the villi. Some of these patients responded promptly to therapy with vitamin B_{12}, and others to folic acid. A gluten-free diet was given to nine patients, and six of them improved considerably, but one patient who failed to improve on the diet after nine months, improved with steroid therapy. A further four patients had normal villi on histological examination although anæmia and diarrhœa were present and the clinical diagnosis of idiopathic steatorrhœa had been made. In all four parenteral administration of iron relieved the abdominal symptoms.

It is suggested that the first group with the abnormal villi be called cœliac disease or adult cœliac disease, as the case may be, but that the second group be regarded as including more than one etiology. The third group is at present still under investigation.

JAUNDICE ASSOCIATED WITH NORETHANDROLONE THERAPY

Obstructive jaundice secondary to the administration of drugs has occurred in association with the use of several compounds of diverse chemical structure. Among these, chlorpromazine and methyltestosterone have received the most attention. Norethandrolone (Nilevar), a potent anabolic agent derived from methyltestosterone, has been incriminated as the cause of obstructive jaundice in two patients. Shaw and Gold (*Ann. Int. Med.*, 52: 428, 1960) report a third case of obstructive jaundice associated with the use of this steroid.

A white woman 60 years of age, with a diagnosis of multiple myeloma, received 2300 mg. of norethandrolone orally over a period of 109 days. Seven days before the end of the planned course of therapy, jaundice with pruritis appeared, together with loss of weight and dark urine. Physical examination was not remarkable with the exception of slight enlargement of the liver, which was not tender. Studies of liver function produced evidence of obstructive jaundice with elevated values for direct bilirubin, alkaline phosphatase and cholesterol. A liver biopsy demonstrated biliary stasis in the bile canaliculi and ductules of a character compatible with intrahepatic obstruction. Because the patient's health was deteriorating, a course of prednisone was prescribed. Chemical and clinical improvement was rapid.

The findings in this case are similar to those reported for jaundice due to methyltestosterone. Norethandrolone is chemically similar to methyltestosterone, and has several metabolic similarities. It is evident that the substitution of an alkyl group in the 17-position of the testosterone nucleus altered the hepatic metabolism of the steroid in a manner that rendered intrahepatic cholestasis a possibility.

TREATMENT OF HYPERTENSIVE CARDIOVASCULAR DISEASE BY SALT-FREE DIET OR DIURETICS

The development and wide use of orally effective diuretics, such as chlorothiazide, has raised the question once again of the importance of sodium in maintaining the hypertensive state. Dustan of Cleveland discusses the antihypertensive effects of low-sodium diet and of diuretics (*J. A. M. A.*, 172: 2052, 1960). A similarity between the two approaches to therapy is obvious. Both cause diuresis and saluresis. Both decrease plasma and extracellular fluid volumes and total exchangeable sodium. Furthermore, not only do both decrease arterial pressure but they also increase the response to antihypertensive drugs. Dustan believes that this suggests that oligæmia induced in the early phases of either low-sodium dietotherapy or diuretic therapy is responsible for the hypertensive effects of these regimens. Whether this mechanism is operative during long-term therapy will have to await further observations.

(Continued on advertising page 26)

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GENERAL HOSPITALS IN CANADA APPROVED BY THE CANADIAN MEDICAL ASSOCIATION FOR JUNIOR (1ST YEAR) INTERN TRAINING

Name of hospital	*Location*	*Beds (excluding bassinets)*	*Teaching beds*						*University affiliation***	*Number of junior interns accepted*	*Monthly stipend*
			Public ward beds					*Private and semi-private*			
			Medicine	*Surgery*	*Obstetrics*	*Pædiatrics*	*Other*				
British Columbia											
Royal Columbian Hospital	New Westminster	434	136	138	34	48	12	66	no	12	$200
St. Paul's Hospital	Vancouver	551	35	56	31	57	105	267	U.B.C.	20	175
St. Vincent's Hospital	Vancouver	193	36	36	37	30	—	—	no	4	100
Vancouver General Hospital	Vancouver	1282	135	265	29	154	258	399	U.B.C.	54	152
Royal Jubilee Hospital	Victoria	444	101	86	30	41	61	125	no	8	175***
St. Joseph's Hospital	Victoria	444	166	165	42	71	—	—	no	10	175***
Alberta											
Calgary General Hospital*	Calgary	743	146	220	59	84	39	195	no	20	100
Holy Cross Hospital	Calgary	338	45	58	32	50	44	109	no	8	100***
Edmonton General Hospital	Edmonton	371	109	70	24	49	10	109	U.A.	10	125
Misericordia Hospital	Edmonton	342	52	60	28	51	—	151	U.A.	8	100***
Royal Alexandra Hospital	Edmonton	729	168	82	104	115	100	140	U.A.	23	100***
University of Alberta Hospital	Edmonton	1078	226	268	32	139	211	202	U.A.	40	100
Saskatchewan											
Moose Jaw Union Hospital	Moose Jaw	244	62	39	11	20	20	92	no	4	200
Regina General Hospital	Regina	739	143	229	26	135	81	125	U.S.	12	125
Regina Grey Nuns' Hospital	Regina	471	121	166	24	50	—	110	U.S.	20	200
St. Paul's Hospital	Saskatoon	277	56	74	12	32	26	77	U.S.	7	225
Saskatoon City Hospital	Saskatoon	345	86	79	18	36	53	72	U.S.	12	225
University Hospital	Saskatoon	530	137	140	78	69	106	—	U.S.	16	100
Manitoba											
St. Boniface General Hospital*	St. Boniface	640	97	129	26	85	83	220	U.M.	26	110
Grace Hospital	Winnipeg	240	32	46	26	8	—	128	U.M.	6	175
Misericordia Hospital	Winnipeg	410	61	119	27	28	—	175	no	8	150
Winnipeg General Hospital*	Winnipeg	830	90	70	24	8	52	354	U.M.	32	110
Ontario											
McKellar General Hospital	Fort William	416	76	54	8	50	—	196	no	9	200
Hamilton General Hospital	Hamilton	1168	52	52	19	33	160	852	no	26	150
St. Joseph's Hospital	Hamilton	479	44	46	26	30	94	239	no	16	175
Hôtel-Dieu Hospital	Kingston	293	59	23	15	20	—	176	Q.U.	10	200***
Kingston General Hospital	Kingston	473	97	92	14	57	20	193	Q.U.	18	180
Kitchener-Waterloo Hospital	Kitchener	426	38	34	24	58	110	162	no	9	250
St. Joseph's Hospital	London	437	71	69	18	40	41	198	W.O.	17	150***
Victoria Hospital	London	877	75	65	15	40	42	630	W.O.	30	140
Oshawa General Hospital	Oshawa	342	59	59	25	—	198	All	no	12	250††
Ottawa Civic Hospital	Ottawa	873	127	210	22	80	19	415	no	35	100
Ottawa General Hospital	Ottawa	622	80	120	24	80	22	296	U.O.	12	175
Hôpital St-Louis Marie de Montfort	Ottawa	229	20	20	24	40	4	30	U.O.	6	150
General Hospital of Port Arthur	Port Arthur	306	61	44	28	41	22	91	no	4	250***
St. Joseph's General Hospital	Port Arthur	198	44	27	7	40	—	80	no	4	200
St. Catharines General Hospital	St. Catharines	360	55	60	11	49	60	125	W.O.	6–8	250
St. Thomas-Elgin Hospital	St. Thomas	371	32	24	16	19	84	182	no	6	175
St. Joseph's Hospital	Sarnia	347	—	—	—	45	—	—	no	6	200
Sarnia General Hospital	Sarnia	247	35	29	8	46	10	119	no	6	215†
Sudbury General Hospital	Sudbury	309	18	36	25	60	22	148	no	8	250
New Mount Sinai Hospital	Toronto	337	36	36	20	10	18	217	no	20	100***
St. Joseph's Hospital	Toronto	507	72	97	26	29	—	283	no	21	125
St. Michael's Hospital	Toronto	830	196	184	30	—	28	392	U.T.	33	70
Toronto East General and Orthopædic Hospital	Toronto	649	70	93	25	20	—	441	no	23	100
Toronto General Hospital*	Toronto	1680	298	394	58	—	151	779	U.T.	40	70.8
Toronto Western Hospital*	Toronto	650	115	115	29	14	23	306	U.T.	27	150
Wellesley Hospital	Toronto	233	52	38	15	—	21	109	U.T.	8	137
Women's College Hospital*	Toronto	286	59	21	49	4	25	128	U.T.	6	75***
Grace Hospital	Windsor	219	19	25	21	35	—	118	no	4	300
Hôtel-Dieu of St. Joseph	Windsor	382	88	93	30	53	8	110	no	8	300***
Metropolitan General Hospital	Windsor	311	46	46	4	41	44	130	W.O.	4	300
Scarborough General Hospital	Scarborough	187	22	41	15	8	—	101	no	8	235‡
Quebec											
Hôtel-Dieu Saint-Vallier*	Chicoutimi	772	60	60	10	30	99	513	U.L.	30	40
Hôpital du Sacré Cœur	Montreal	735	34	33	12	10	382	264	U.M.	14	40
Hôpital St-Luc	Montreal	413	93	167	15	5	—	132	M.	20	90
Hôtel-Dieu de Montréal*	Montreal	750	107	130	—	—	—	480	M.	22	40
Jewish General Hospital	Montreal	382	57	45	30	21	43	186	McG.U.	16	65
Maisonneuve Hospital	Montreal	511	43	40	6	24	30	368	M.	19	40
Montreal General Hospital*	Montreal	721	142	142	43	—	66	328	McG.U.	48	65
Notre-Dame Hospital	Montreal	690	65	60	10	60	85	410	M.	30	40
Queen Elizabeth Hospital*	Montreal	273	32	36	20	—	—	185	McG.U.	9	100
Royal Victoria Hospital	Montreal	907	60	130	48	37	64	568	McG.U.	32	65
Reddy Memorial Hospital****	Montreal (Westmount)	139	10	18	14	2	5	90	McG.U.	7	100
St. Mary's Hospital	Montreal	301	29	44	12	30	16	57	no	16	150
Hôpital de l'Enfant-Jésus*	Quebec	502	—	—	—	—	—	—	U.L.	20	25
Hôpital du Saint-Sacrément*	Quebec	302	24	24	—	15	—	239	U.L.	18	25
Hôpital St-Francois-d'Assise*	Quebec	320	36	42	22	30	60	130	U.L.	9	25
Hôtel-Dieu de Québec*	Quebec	310	26	40	8	10	—	226	U.L.	18–20	25
Jeffery Hale's Hospital	Quebec	150	12	12	4	12	—	110	U.L.	4	25
Hôpital Général St-Vincent-de-Paul	Sherbrooke	269	36	39	8	38	5	143	U.L. & M.	6	50
Hôtel-Dieu de Sherbrooke	Sherbrooke	337	75	70	22	52	—	118	U.L.	6	50
Sherbrooke Hospital	Sherbrooke	150	54 combined		8	18	—	70	McG.U.	6	150
Hôpital St-Joseph	Trois-Rivières	237	10	20	—	34	—	173	U.L.	8	25
Hôpital Général de Verdun	Verdun	420	62	46	10	42	—	136	M.	7	40
New Brunswick											
Victoria Public Hospital	Fredericton	169	47 combined		7	26	13	76	D.U.	3	75
Moncton Hospital	Moncton	210	16	16	8	42	—	128	D.U.	5	150
Saint John General Hospital	Saint John	522	65	64	22	52	54	265	D.U.	14	100
Nova Scotia											
Halifax Infirmary*	Halifax	223	27	32	29	—	—	135	D.U.	9	75
Victoria General Hospital*	Halifax	558	112	131	—	—	105	202	D.U.	48	75
Newfoundland											
St. John's General Hospital*	St. John's	452	48	108	—	53	139	54	D.U.	15	100

*Hospitals having arrangements with other hospitals for part of rotation program.

Parent hospitals	*Hospitals supplementing intern training*
Calgary General Hospital, Calgary, Alta.	Salvation Army Grace Maternity Hospital, Calgary (Antenatal Clinics)
St. Boniface General Hospital, St. Boniface, Man.	Children's Hospital, Winnipeg (Pædiatrics) D.V.A., Winnipeg (Medicine)
Winnipeg General Hospital, Winnipeg, Man.	Children's Hospital, Winnipeg (Pædiatrics)
St. Michael's Hospital, Toronto, Ontario	Hospital for Sick Children, Toronto (Pædiatrics)
Toronto General Hospital, Toronto, Ontario	Hospital for Sick Children, Toronto (Pædiatrics)
Toronto Western Hospital, Toronto, Ontario	Hospital for Sick Children, Toronto (Pædiatrics)
Women's College Hospital, Toronto, Ontario	Toronto East General and Orthopædic Hospital (Pædiatrics)
Hôtel-Dieu de Montréal, Montreal, Quebec	Hôpital Ste-Justine, Montreal (Obstetrics and Pædiatrics)
Hôtel-Dieu de Québec, Quebec, Quebec	Hôpital Laval, Quebec (Medicine and Surgery) Hôpital de la Miséricorde, Quebec (Obstetrics) Crèche St-Vincent-de-Paul, Quebec (Pædiatrics) Hôpital Général St-Vincent-de-Paul, Sherbrooke (Surgery and Obstetrics)
Hôtel-Dieu St-Vallier, Chicoutimi, Quebec	Hôtel-Dieu Notre-Dame-de-l'Assomption, Jonquière (General)
Hôpital de l'Enfant-Jésus, Quebec, Quebec	Hôpital St-Michel-Archange, Quebec (Psychiatry and Neurology) Hôpital Laval, Quebec (Medicine and Surgery) Hôpital de la Miséricorde, Quebec (Obstetrics)
Hôpital du Saint-Sacrement, Quebec, Quebec	Hôpital de la Miséricorde, Quebec (Obstetrics) Hôpital St-François-d'Assise, Quebec (Obstetrics and Pædiatrics) Hôpital Ste-Foy, Quebec (Medicine and Surgery) Hôpital St-Michel-Archange, Quebec (Psychiatry and Neurology) Crèche St-Vincent-de-Paul, Quebec (Pædiatrics)
Hôpital St-François-d'Assise, Quebec, Quebec	Hôpital St-Michel-Archange, Quebec (Psychiatry and Neurology)
Montreal General Hospital, Montreal, Quebec	Catherine Booth Mothers' Hospital, Montreal (Obstetrics) Montreal Children's Hospital, Montreal (Pædiatrics) Charlotte Memorial Hospital, Charlotte, North Carolina (Medicine, Surgery, Obstetrics and Pædiatrics) King Edward VII Memorial Hospital, Bermuda (General) Royal Edward Laurentian Sanatorium, Ste-Agathe (Chest Diseases) General Hospital, Sherbrooke (General) Verdun Protestant Hospital (Psychiatry)
Queen Elizabeth Hospital, Montreal, Quebec	The Montreal Children's Hospital (Pædiatrics)
Halifax Infirmary, Halifax, N.S.	Children's Hospital, Halifax (Pædiatrics) St. Elizabeth Hospital, North Sydney (Pædiatrics)
Victoria General Hospital, Halifax, N.S.	Halifax Children's Hospital, Halifax (Pædiatrics) Grace Maternity Hospital, Halifax (Obstetrics)
St. John's General Hospital, St. John's, Newfoundland	Grace Hospital, St. John's (Obstetrics)

***University abbreviations used in list of approved hospitals.*

U.B.C.—University of British Columbia
U.A. —University of Alberta
U.S. —University of Saskatchewan
U.M. —University of Manitoba
Q.U. —Queen's University
W.O. —University of Western Ontario
U.O. —University of Ottawa
U.T. —University of Toronto
U.L. —Laval University
M. —University of Montreal
McG.U.—McGill University
D.U. —Dalhousie University

***Living out allowance in addition to salary.
****Credit for additional beds given for Home Care Service.
†Free meals.
‡235 then 250.
††Canadian graduates, foreign graduates, $200.

MEDICAL MEETINGS

INTERNATIONAL SOCIETY OF TROPICAL DERMATOLOGY

The Inaugural Meeting of the International Society of Tropical Dermatology was held on May 10 at the Caspary Hall of the Rockefeller Institute in New York City. After a brief opening ceremony Dr. René Dubos welcomed the assembly on behalf of Dr. Detlow Bronk, President of the Institute, emphasizing the importance of the new organization and expressing his gratification that the seat of the Inaugural Meeting was the Rockefeller Institute, more especially since Professor Aldo Castellani began his career in the field of tropical medicine at the same time that the Rockefeller Institute was founded.

"Elephantiasis tropica and elephantiasis nostra" was the topic of Professor Castellani's presidential address. Many decades of experience, both in the tropics and temperate zones (England, Italy, Scotland, and Switzerland), were recounted. Emphasis was placed

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upon the similarity of the clinical symptomatology of elephantiasis tropica and elephantiasis nostra. While filariæ may act as a primary irritating and traumatic agent in tropical elephantiasis, the real cause of elephantiasis is a bacterial infection, similar to what has been recognized to be the cause of elephantiasis in non-tropical countries. The repeated isolation of a minute coccus, called *Micrococcus metamyceticus*, on special culture media was reported as evidence of a causal relationship between the diseases. Futhermore, the successful amelioration of these diseases with a vaccine prepared from this microorganism was an additional support of his viewpoint.

The Secretary-General delivered a short address informing the members that 50 countries are represented in this organization with almost 1300 charter members. The date and place of the First International Congress were discussed, and it was considered most likely that this meeting would be held either in Mexico City or Rio de Janeiro in 1962 or in 1963.

OBITUARIES

DR. LOUIS-PHILIPPE BEAUDOIN of Hawkesbury, Ont., aged 68, died June 9. Born in Broughton, Que., he received his medical degree from Laval University in 1919 and specialized in surgery in New York. He returned to Canada and practised in Hawkesbury and district for 41 years.

Surviving Dr. Beaudoin are his widow, three sons and four daughters.

DR. ERNEST VICTOR FREDERICK, 79, Toronto physician and surgeon, died July 12 at his home in Scarborough, Ont.

Born in Campbellford, Ont., he graduated from the University of Toronto in 1903.

Dr. Frederick took postgraduate studies in Europe and practised in Peterborough, Ont., and in Hollywood and Reno. He later returned to Toronto and was a surgeon on the staff of the Toronto General and East General Hospitals. For some time he lived at Centre Island and tended patients from Hanlan's Point to Ward's Island.

Dr. Frederick is survived by his widow.

DR. AUGUSTE LEBŒUF, aged 78, died July 2 in Deschaillons, Que. Born in Deschaillons, he graduated from Laval University in 1907, and practised for several years in Deschaillons and served overseas in the R.C.A.M.C. in the First World War.

He was assistant superintendent at Lake Edward Sanatorium and a pioneer in the fight against tuberculosis.

DR. HENRY W. MILLER, 86, retired neurologist and psychiatrist, died in Brewster, New York, June 23.

Born in Orillia, Ont., he graduated from the University of Toronto in 1895. He held posts at hospitals in Waverly and Taunton, Mass., Cook County Hospital in Chicago, St. Elizabeth's Hospital in Washington, Maine State Hospital, Augusta, and Vanderbilt Clinic and Neurological Institute at Columbia Presbyterian Center, New York.

Dr. Miller is survived by one son.

PROVINCIAL NEWS

ALBERTA

Dr. K. J. Williams, who for the past year has been associate medical superintendent of the Royal Alexandra Hospital in Edmonton, has resigned from that position to take over the duties of medical director of St. Joseph's Hospital in Hamilton, Ontario.

At a recent public meeting in Valleyview, a community in the Peace River area, Dr. J. Donovan Ross, Alberta's Minister of Health, discussed the government's policy on the provision of hospital facilities for areas that have none. The meeting had been organized by the Valleyview Chamber of Commerce to promote the building of a hospital to serve their community. The representation of the residents pointed out that Valleyview is on the main highway serving the Peace River country and has highways radiating out from it. The nearest hospital to the south is 135 miles away and to the west 68 miles away, leaving over 200 miles of busy arterial highways without a doctor or a hospital. A district nurse whose job is preventive medicine and first aid serves the area. Dr. Ross held out no hope for a hospital in the near future. In order to approve a hospital he said, there must first be established a medical service; as well, the population in the area is not sufficient to warrant a hospital, as nothing smaller than a 25-bed hospital is approved by the Department of Health.

Plans are being processed for a new Charles Camsell Hospital in Edmonton. The present hospital which was opened in 1946 is inadequate to serve as it does Indians from Alberta, North West Territories and the Yukon, and Eskimos and Metis from the Northwest Territories. Eighty-five thousand dollars has been provided this year for initial planning of the structure which, it is estimated, will cost $8,500,000, and is expected to contain 450 beds.

The North American Association of Alcoholism Programs will hold its annual meeting and conference at the Banff School of Fine Arts on September 25 to 30, 1960. Inquiries should be directed to J. George Strachan, Chairman, N.A.A.A. Program Committee, 9910-103 Street, Edmonton, Alberta.

Work has started on the $755,000 addition to the Good Samaritan Hospital in Edmonton. This hospital, which is operated by the Lutheran Home Society, accommodates 96 chronic patients. Federal and provincial grants make up a large part of the capital available.

The Edmonton Rehabilitation Society's new downtown treatment centre was opened last month with Premier Manning officiating. Also in attendance was Lieutenant Governor Page and several members of the provincial cabinet. In addition to the many phases of rehabilitation, the centre houses Goodwill Industries Inc., where handicapped persons renovate used furniture, clothing and electrical appliances for sale and on order.

W. B. PARSONS

The white areas on this aerial view of the Hospital for Sick Children, Toronto, illustrate where construction will be undertaken as part of the $8 million building program beginning in November. The first additions to be made since the Hospital was opened in 1951, the building program will provide a ten-storey structure for the north back wing (foreground), add three floors to the centre back wing and replace the present structures adjacent to the back south wing with a new three-storey building.

ONTARIO

Planned Expansion of the Hospital for Sick Children —Toronto

The Toronto Hospital for Sick Children will begin an $8 million expansion program next November, as announced on July 19 by J. Grant Glassco, Chairman of the Board of Trustees. About $1,700,000 of this amount is anticipated from capital grants. For the remainder of the expenditure the Hospital will turn to its many friends who have supported it so generously in the past. A public appeal for funds will not be made.

This will be the first addition to the building since the hospital was opened in 1951. Three main objectives of the program are: the provision of better facilities for research; the development of highly specialized facilities to meet medical needs that have developed since the hospital was constructed; and the streamlining of existing buildings and services to adjust to the increased number of patients. The need for improved facilties for study and investigation of disorders of infants and children is emphasized by the fact that while many nations have a neonatal mortality of 14 or less per 1000 live births, the neonatal mortality rate in Canada, despite our high living standards, is 24 per 1000 live births.

While the addition will provide 176 new beds, bringing the total to 823 children's beds, the Hospital's board believes that this lies within their policy announced in 1957, stating that other hospital authorities and medical groups must now give consideration to the problems of providing additional hospital facilities for the growing child population in Toronto and throughout the province. The board observed that increasing demands are being placed upon the Hospital for Sick Children and warned that the current addition of beds will bring it close to its ultimate patient maximum.

Initial construction plans will be put into effect with a call for tenders in November 1960. Estimated date of completion for the total program by planned stages is December 1964.

The new building plan involves the following features: (1) Expansion of the Emergency Department by addition of new rooms for examination and treatment, two major and three casualty operating rooms, emergency x-ray facilities, and an observation ward. (2) Additional operating rooms for general surgery, cardiac

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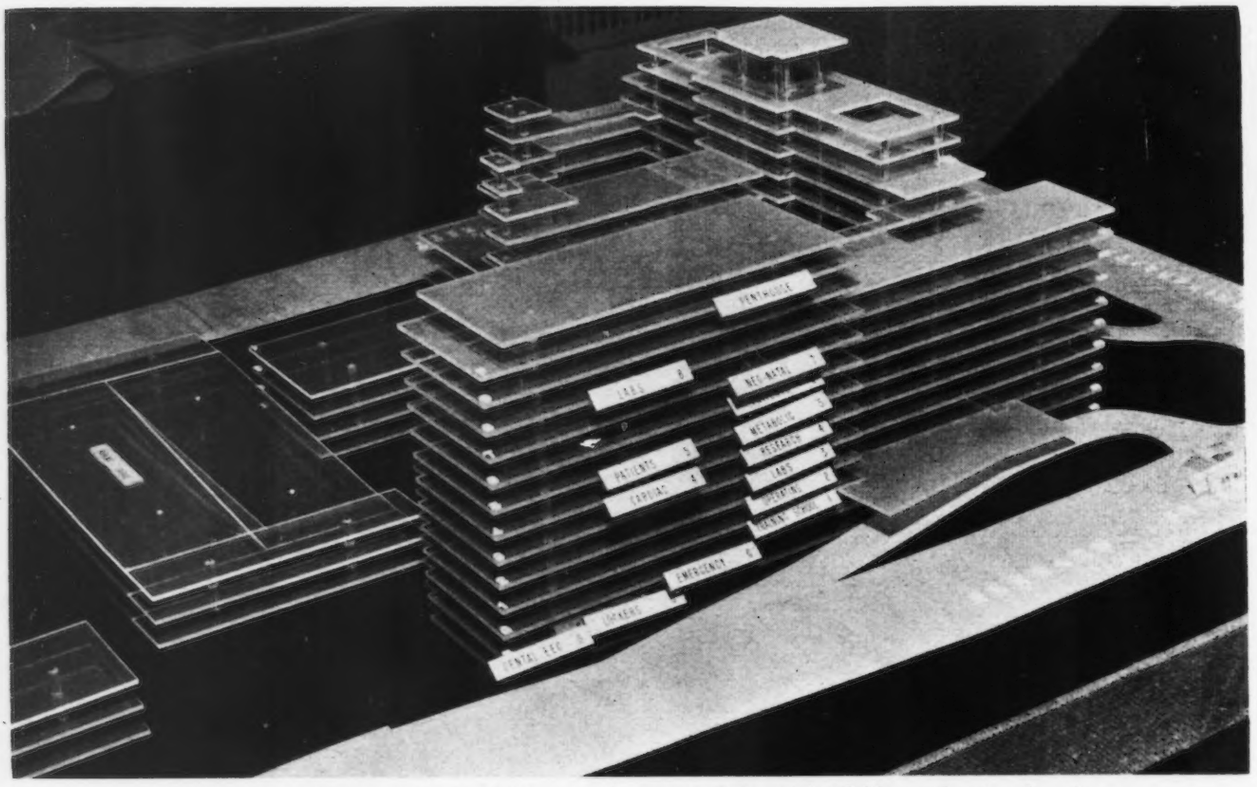

Plastic model of the proposed addition to the Hospital for Sick Children, showing location of the various departments.

and neurosurgery with a monitor room connecting the cardiac and neurosurgery theatres, housing the complicated electronic recording equipment used during surgery. (3) New research and laboratory facilities for investigation of disorders of the liver, bone, kidney, cardiovascular and endocrine systems, virology and biochemistry research laboratories, expanded laboratory facilities for routine hospital work, and a cardiorespiratory physiology laboratory. (4) Additional bed space for 132 medical and surgical patients. (5) An isolation wing for 44 medical and surgical cases. (6) New laundry and orthopædic shop, and (7) internal changes to improve facilities of the admitting office, recovery rooms and out-patient clinics.

Dr. Charles H. Best was elected an honorary president of the American Diabetes Association at its 20th annual meeting at Miami Beach. He is a past president of the Association.

Dr. U. J. Durocher, Windsor, has been honoured by his alma mater, Wayne State University College of Medicine, with a golden anniversary graduation diploma. He was one of the class of 39 graduating in 1910. Eleven members of this class are alive and three are practising, two in Detroit and one in Windsor.

Dr. David Robertson, department of pathology, Queen's University, has been awarded a $9000 fellowship by the Canadian Cancer Society. He will take two years' training in neurology and neuropathology at the University of Toronto; at the National Hospital, London; and at Cornell Medical Center, New York. Dr. Robertson is a native of Weyburn and a Queen's graduate of 1955.

The executive of the Essex County Medical Society has arranged a meeting of the Association of French Speaking Doctors to be held in the Cleary Auditorium, Windsor, September 20-23. There will be no registration fee. A ladies' program has been arranged and there will be banquets and cocktail parties. A translating service similar to that used at the United Nations will be provided. There are about 24 French-speaking doctors in the Essex County Medical Society.

Last year the Thunder Bay Nurses' Registry emergency communication centre handled 235,000 telephone calls. A nurse is always on duty and can call all hospitals, 68 doctors, 25 or 30 registered nurses, dentists, druggists, police and fire departments and numerous welfare agencies, when necessary.

The registry is a non-profit organization and operates each year with a deficit. Fort William gives a yearly grant of $125 and Port Arthur one of $75. The annual cost of operating the registry is about $12,000. Most of this cost comes from subscription rates paid by doctors, nurses, druggists and ambulance services.

During the $60,000 fire at Peterborough Civic Hospital on May 5, all hospital personnel carried out the prearranged fire drill without hesitation, and more than 120 patients were taken from the hospital without injury. The fire destroyed the staff cafeteria and filled the wards with smoke.

At a dinner of the Variety Clubs International held in the Royal York Hotel, Toronto, Dr. Jonas Salk, University of Pittsburgh, was presented with the International humanitarian award. Dr. Salk was the 21st person to achieve the award, which was established in 1939.

LILLIAN A. CHASE

To help relieve the bed shortage in Hamilton hospitals, 30 beds have been set aside in the Evel Building of Mountain Sanatorium. The fourth floor of the sanatorium was opened for general surgery on March 6. Plans are under way for the opening of the Chedoke Hospital in about a year. The Evel Building will be a temporary base for the hospital's operations, pending completion of renovations in the Wilcox Building, its permanent location.

The Parry Sound General Hospital recently opened an 86-bed addition, which involved an expenditure of $600,000 for the building plus $250,000 for furniture and equipment.

The 52-year-old Welland County General Hospital was replaced recently with the opening of a $4,150,000 building. The structure contains 257 adult beds plus 51 bassinets. The hospital has been designed so that its capacity can be increased to approximately 450 beds by the addition of a south wing.

There are six operating rooms, located on the third floor. Two are devoted to and equipped for general surgical work, while the remainder will be used for orthopædic, neurological, ear, nose and throat, and ENT combined with general work. Also, two emergency operating rooms are located in the emergency department on the ground floor.

An artificial kidney will be made available for the first time to Northern Ontario hospitals through the generosity of the Sudbury Lions Club. The Lions presented the machine to the Sudbury and District Medical Association at a recent ceremony held in the Sudbury General Hospital. The machine will be kept at the Sudbury-Algoma Sanatorium, but it can be transported readily to any hospital in the district.

At the Princess Margaret Hospital in Toronto, a team consisting of Dr. H. E. Johns, Dr. J. Cederlund and Mr. Daniel Rotenberg are working on the development of a body scanning apparatus which it is hoped will assist in the location of malignant metastatic lesions by means of a detector sensitive to the emanations of radioactive compounds. Difficulties in devising apparatus for tumour detection capable of widespread application are presented by the problems of finding radioactive materials which are selectively deposited in malignant cells and not in normal tissue constituents, and at the same time have a half-life sufficiently short to avoid widespread damage to normal cells. The equipment developed to date is still in the experimental stage.

Dr. Norman S. Shenstone, an outstanding Toronto surgeon, was recently honoured by members of the Medical Board and Board of Directors of St. John's Convalescent Hospital, Newtonbrook, upon his retirement after 16 years as chairman of the Medical Board of the Hospital. He is succeeded by Dr. F. P. Dewar.

Dr. Shenstone was presented with his portrait, which was unveiled by Reverend Sister Beatrice, former Superintendent of the hospital. The portrait was painted by the Canadian artist Cleeve Horne, and will hang in the main entrance to the hospital.

Dr. R. I. Harris, representing the Medical Board, praised Dr. Shenstone's work, especially his contributions to the field of chest and cardiac surgery. Dr. Shenstone has been associated with St. John's for the past 50 years and will continue as a member of the active staff.—*Hospital Highlights* (Ontario Hospital Association, July 1960).

QUEBEC

A private, closed-circuit television show designed to provide a large audience with a twice-life-sized view of surgical operations was exhibited at the recent clinical program of the Continental Gynecologic Society in the Montreal General Hospital, attended by more than 70 gynæcologists from Canada and the United States. The pictures were viewed on four 23-inch screen sets in the hospital auditorium, the sets being linked by cable with the camera equipment mounted in the operating room gallery. To provide the audience with a magnified, clear view, the direct operating area alone was pictured by means of a zoom-type telephoto lens. For each of the three types of operation illustrated, the surgeon wore a compact lapel microphone, providing a running commentary on the operative technique, while a two-way communication system with a microphone in the auditorium allowed members of the audience to put their questions to the surgeon in the operating theatre.

CANADIAN ARMED FORCES

National Defence photo

Major D. Howell Bevan-Jones

Major D. Howell Bevan-Jones, 43, Royal Canadian Army Medical Corps, of Chester, N.S., commanding officer of the Camp Gagetown Station Hospital and consultant psychiatrist for the New Brunswick area for the past two years, will take up a new appointment in mid-August with the Canadian Forces' Hospital in Halifax. A native of Llandile, Wales, Major Bevan-Jones served with the Royal Navy during the Second World War. He was appointed a medical officer in the Canadian Army in 1951 and since that time has served in Quebec, Halifax, Korea and Camp Gagetown.

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ABSTRACTS *from current literature*

MEDICINE

An Accurate Method of Determining Sedimentation Rate.

J. B. DAWSON: *Brit. M. J.*, 1: 1697, 1960.

The various methods of estimating the erythrocyte sedimentation rate employed in the Edinburgh Hospitals have been investigated by the author and have been found wanting. He reviews the history and the various methods that have been used in the past to determine the E.S.R. and presents his own experimental work to support a new method which he believes to be far superior to all the previous ones. It employs sequestrene as an anticoagulant and uses a suspended Westergren pipette instead of the static pipette. He believes that the normal range should be higher by a further 5 mm. and that within this new range, low readings such as 1 and 2 mm. should be considered as abnormal. In adults over 50 years of age, higher values may be normal, and in pregnancy the normal range should be considered up to 45 mm. in one hour. Dawson states that sequestrene (obtainable as the di-potassium or di-sodium salt from the General Pharmaceutical Company Ltd., Judex Works, Sudbury, Middlesex) acts as an anticoagulant by chelating the calcium ions of the blood and forming a soluble non-ionized salt.

Dawson prefers the Westergren pipette because it requires only about 1 ml. of blood, and because it is in wide general use. The Wintrobe pipette should not be used. W. GROBIN

Phonocardiography in Pulmonary Stenosis: Special Correlation between Hæmodynamics and Phonocardiographic Findings.

E. G. DIMOND AND A. BENCHIMOL: *Ann. Int. Med.*, 52: 145, 1960.

The auscultatory and phonocardiographic findings were studied in 56 patients with pulmonic stenosis proved by cardiac catheterization. These included 21 cases of isolated pulmonic stenosis, 30 cases of pulmonary stenosis associated with ventricular septal defect, and five cases of pulmonary stenosis with interatrial communication. Isolated pulmonic stenosis usually produced a widely split second sound, with a diminished or absent pulmonic component, prolongation of right ventricular systole, and a systolic murmur going through the aortic component of the second sound. These findings correlated well with the degree of right ventricular hypertension. In cases of tetralogy of Fallot, the time of accentuation of the systolic murmur did not show a significant difference from the murmur of isolated pulmonic stenosis, and the murmur usually stopped at the aortic component of the second sound. However, in five cases of proved tetralogy of Fallot, the murmur went through the aortic component of the second sound. The presence of giant *a* waves in the venous tracing suggested a diagnosis of moderate to severe pulmonary stenosis with intact ventricular septum. The presence of a systolic ejection click was helpful in the evaluation of the degree of stenosis, but it was recorded only 14 times in the whole series, and it was present in only mild and moderate stenosis. S. J. SHANE

Effect of Desiccated Thyroid on the Hyperlipoproteinæmia of Xanthoma Tendinosum.

B. STRISOWER *et al.*: *Am. J. M. Sc.*, 239: 71, 1960.

A group of 20 patients with the s_f0-20 hyperlipoproteinæmia characteristic of xanthoma tendinosum was studied for an average of two years. Twelve of the 20 patients had the tendon lesions characteristic of xanthoma tendinosum. The 20 patients were given desiccated thyroid (maximum average daily dose, 6 grains) for an average of 36 weeks. Both the s_f0-12 and s_f12-20 low density lipoprotein concentrations were promptly and significantly reduced by the administration of desiccated thyroid, but no changes in the s_f20-100 or s_f100-400 low density lipoprotein concentrations were observed. The atherogenic index and the serum cholesterol concentration were significantly reduced by thyroid administration. It is suggested that reduction of the elevated s_f0-20 serum lipoprotein concentrations in xanthoma tendinosum would lower very considerably the morbidity and mortality from coronary heart disease in these patients. S. J. SHANE

Aneurysms of the Abdominal Aorta and Fever.

F. W. TEN EYCK *et al.*: *Proc. Staff Meet. Mayo Clin.*, 35: 1, 1960.

This is a case report of an atherosclerotic abdominal aneurysm which was infected and subsequently ruptured. It is emphasized that the presence of bacterial infection should be suspected in patients with abdominal aneurysms and fever. Positive blood cultures would lend support to such a suspicion. Leukocytosis and anæmia are common in patients with ruptured aneurysms, but a significant elevation of temperature is uncommon.

The reported incidence of infected arteriosclerotic aneurysms depends on the clinician's awareness of the condition and the pathologist's utilization of special methods, including culture and Gram stains of the lesion, at the time of necropsy.

Once an arteriosclerotic aneurysm becomes infected the likelihood of rupture is high. Prophylactic antibiotic therapy should be considered whenever bacteræmia is likely to develop in patients with aneurysms of large vessels. S. J. SHANE

Environmental Factors in Coronary Heart Disease. An Epidemiological Study at Agra (India).

K. S. MATHUR: *Circulation*, 21: 684, 1960.

Various factors relating to coronary heart disease were investigated in two groups of patients: 553 patients with clinical coronary heart disease and 1056 persons selected in a field survey of the general population of Agra, India.

The incidence of coronary disease was higher in the urban population, in the upper socio-economic classes, and in persons with the highest amounts of total dietary fat and highest percentage of calories from dietary fat. Values for total serum cholesterol, serum phospholipid, and cholesterol/phospholipid ratio were higher in the coronary group.

Finally, it seems that physical activity was less and emotional stress and strain greater in the patients with coronary disease.

No relation between coronary disease and smoking or alcohol was established. S. J. SHANE

Malignant Disease in the Aged: Review of 16 Years' Experience in a Modern Medical Facility for the Elderly.

E. ELIAS AND G. J. LESNICK: *Am. J. M. Sc.*, 239: 554, 1960.

The incidence and results of management of malignant disease in a home for the aged are reviewed. Evidence of malignant diseases prior to death or at postmortem examination was found in 16% of patients dying in a home. Three-fourths of them died of malignancy. Tumours of the gastro-intestinal tract comprised 49.6%; the genito-urinary tract, 16%; the lungs, 11.9%, and the breast, 10.9%. Diagnosis was established before death in two-thirds of the patients. In a little over one-third of the patients, diagnosis was established early enough to permit treatment with some hope of success. Treatment gave significant palliation in two-thirds of those treated and resulted in cure of the disease in one-fourth.

This study indicates the need for simple screening tests, such as blood and stool examination, and frequent weight measurements in older individuals. The excellent response to therapy in many of the patients emphasizes the value of early aggressive therapy even at advanced ages. S. J. SHANE

Pulmonary Artery Wedge Catheter Position as a Site for Injection of Indicator Substance.

G. G. ROWE *et al.*: *Circulation*, 21: 724, 1960.

The pulmonary wedge position is described as a site for injection of indicator-dilution substance because it presents the most central point in the circulation that can be attained during right heart catheterization without actually entering the left side of the heart. Injection at this site furnishes a small "central volume" and produces curves that for the same quantity of dye injected present a sharper peak concentration, a shorter buildup and disappearance time, and therefore a curve the morphology of which is more rigidly set than those obtained by injection at more proximal sites in the right side of the heart. Because the curve is sharper, small recirculation curves on the washout slope are more readily apparent: therefore smaller shunts can be detected during diagnostic cardiac catheterization. S. J. SHANE

Inhalation and Skin Test in the Diagnosis of Asthma Bronchiale.

H. NILSSON AND J. KAUDE: *Dis. Chest*, 37: 535, 1960.

The inhalation test is a valuable investigational method in the diagnosis of bronchial asthma. It can be carried out simply and safely, its results being superior to those of skin tests. Observations based upon 1070 inhalation tests in 200 asthmatics showed that dermal sensitivity corresponds to pulmonary sensitivity in only one-third of cases with positive skin tests. However, in the presence of strong skin reactions the incidence of positive inhalation tests is high. Milder skin reactions are less valuable in selection of allergens for performance of inhalation tests. The highest coincidence in positive skin and pulmonary reactions was found in tests with animal hair extracts. In cases of negative skin test the allergen concerned may induce a positive inhalation test in approximately 21%. The authors suggest that the inhalation test be cautiously employed as an adjunct to skin testing.

S. J. SHANE

Comparative Morbidity and Mortality of Antimicrobially Treated and Untreated Idiopathic Effusion in the Negro.

J. H. SEABURY, J. B. BOBEAR AND J. M. LIBERMAN: *Dis. Chest*, 37: 483, 1960.

In this study, 47 American Negroes with idiopathic pleural effusion were treated with bed rest of variable duration together with therapeutic aspiration; 64% of them developed proved tuberculosis during a minimum follow-up of five years and 21% of them died. During the same period of time and under the same hospital conditions, 45 American Negroes were given chemotherapy, most of which was inadequate by present day standards, and were then followed for a similar period of time. Of this group 17.7% developed postpleuritic tuberculosis, and 4.4% died.

This shows that even inadequate antimicrobial therapy was able to alter significantly postpleuritic morbidity and mortality from tuberculosis. The mortality among the untreated group exceeded the morbidity among the treated. Pleural and thoracic complications of serofibrinous effusion were significantly fewer and less severe after antimicrobial therapy. The data suggest that inadequate antimicrobial therapy may prolong the interval between effusion and relapse in the American Negro. It is the belief of the authors that idiopathic pleural effusion in the American Negro is a particularly serious event, and when it is associated with a positive tuberculin reaction, should be treated as miliary tuberculosis.

S. J. SHANE

Epidemiology of Group-A β-Hæmolytic Streptococci as Related to Acute Rheumatic Fever in Florida. A Six-Year Study.

M. S. SASLAW AND J. M. JABLON: *Circulation*, 21: 679, 1960.

This study discloses that rheumatic fever and rheumatic heart disease are infrequent in Dade County, Florida. However, in this area, β-hæmolytic streptococci are frequently recovered, particularly from the throats of children six to nine years old.

The juxtaposition of these two findings suggests that the relationship between group-A β-hæmolytic streptococci, clinical illness, and rheumatic illness may be different in southern Florida from elsewhere in the United States. S. J. SHANE

Isoniazid Therapy of Primary Tuberculosis in Children.

K. H. K. HSU: *Dis. Chest*, 37: 499, 1960.

Isoniazid was found to be effective in preventing tuberculous complications, thus minimizing the danger of primary tuberculosis, and to be a remarkably effective agent for the treatment of all clinical forms of tuberculosis in children. Since effective therapy is now available, the writer believes that every effort should be made to seek out the infected children by routine tuberculin testing so that they may receive the benefit of isoniazid therapy in the early stage of the infection. Special attention should be given to children under 3 years of age because of the high mortality and morbidity rates of tuberculosis at this age period. In countries where the incidence of tuberculosis is low and where BCG vaccine is not in general use, routine tuberculin testing of young children serves the dual purpose of early treatment of primary tuberculosis in children and the detection of unrecognized tuberculosis in adults. S. J. SHANE

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SURGERY

Deterioration After Mitral Valvotomy.

C. BAKER AND W. E. HANCOCK: *Brit. Heart J.*, 22: 281, 1960.

Two hundred patients, who had undergone mitral valvotomy, were followed up for over five years. Clinical reassessment was made at yearly intervals. The proportion of patients with good results fell from 85% in the first year to 66% in the fifth year, a steady deterioration rate of 5% per annum.

There were three causes of deterioration after valvotomy. The operation may be inadequate and leave some degree of stenosis. Secondly, the valve may restenose. The third cause is made up of a group of factors which include mitral regurgitation, atrial fibrillation, aortic and tricuspid disease, chronic pulmonary disease, pregnancy, bacterial endocarditis, arterial embolism, hypertension, rheumatic activity and a myocardial factor leading to congestive failure. Significant mitral regurgitation, whether surgically produced or pre-existing, produces rapid deterioration. Calcification of the mitral valve prevents satisfactory relief of the stenosis, while making a full opening may increase the regurgitation severely. Onset of atrial fibrillation and deterioration after an initial good result were closely associated. Patients with involvement of the aortic and tricuspid valves had extensive rheumatic heart disease and deteriorated more rapidly. Those with tricuspid lesions had convincing evidence of active rheumatic heart disease even after a successful mitral valvotomy. Severe pulmonary hypertension before operation did not adversely affect the long-term benefits. Persistent bronchitis did not necessarily prevent a good result.

Restenosis occurred in 10 cases and only in patients who had not obtained a satisfactory increase in the mitral valve area. Where complete valvotomy had been performed, no patient deteriorated from restenosis alone. A total follow-up period of 6 to 11 years revealed that 54% still had good results and were without evidence of deterioration. I. H. SHLESER

Surgical Indications in Cholelithiasis.

J. LUND: *Ann. Surg.*, 151: 153, 1960.

A follow-up of 526 patients with gallstones who were not operated upon, 5 to 20 years after the diagnosis was made, is reported from the Copenhagen County Hospital. In some cases the stones were discovered as a chance finding in laparotomies or radiograms performed for unrelated reasons, but in most instances, cholelithiasis was diagnosed on clinical evidence plus non-visualization of the gallbladder. In a few patients negative shadows were seen on the cholecystogram.

Irrespective of the clinical severity of the symptoms and signs of gallbladder disease, at least one-half or one-third of the patients subsequently developed severe symptoms and/or complications. Complications of acute cholecystitis or common duct stone occurred later in one-quarter of this series. This was true of asymptomatic stones too. The mortality of cholelithiasis proved to be at least 2.7% though a number of deaths were undoubtedly avoided by subsequent operations. Over the age of 60, the mortality was over 7.2%. Cancer of the gallbladder occurred in three cases, the risk being under 1%.

Prophylactic cholecystectomy is recommended in all cases of gallstone if the patient is a reasonable surgical risk. The mortality of the primary operation is lower than that for non-operative treatment. The risk of carcinoma is less than the operative mortality of elective cholecystectomy, so that this is not *per se* a reason for the operation, although it is the only effective measure against gallbladder cancer at present.

BURNS PLEWES

Surgical Correction of Coarctation of the Main Pulmonary Artery.

W. B. THROWER, W. H. ABELMANN AND D. E. HARKEN: *Circulation*, 21: 672, 1960.

Two cases of pure congenital coarctation of the main pulmonary artery are reported. One case may be unique in that it represents the first report of an anatomically proved constriction located in the middle third of the main trunk. In this patient, successful open correction under hypothermia was carried out with cinematographic documentation. Results of preoperative and postoperative cardiac catheterization are described.

A recommendation is made for the routine use of open operation for correction of "pure pulmonic valvular stenosis". This allows better correction when the diagnosis is confirmed and flexibility for treatment if this unusual coarctation or constriction of the pulmonary artery is encountered. S. J. SHANE

Intestinal Malrotation and Duodenal Ileus.

J. H. LOUW: *J. Roy. Coll. Surgeons Edinburgh*, 5: 101, 1960.

A proportion of cases of chronic duodenal ileus in children and adults are examples of malrotation with duodenal compression by congenital bands often aggravated by midgut volvulus. It may be necessary to mobilize the colon and duodenum to recognize the causative congenital bands.

Symptoms may be bizarre: dyspepsia, attacks of abdominal pain, flatulence, distension and steatorrhœa, so that there is an impression of deep-seated neurosis. There is a high incidence of duodenal ulceration. Once recognized, the situation is easily remedied surgically. BURNS PLEWES

Blunt Trauma to the Abdomen.

G. L. WATKINS: *A.M.A. Arch. Surg.*, 80: 187, 1960.

During ten years, 141 patients presenting with abdominal injuries caused by blunt trauma were admitted to St. Louis City Hospital. The majority were involved in car accidents and 40% were admitted between 10.00 p.m. and 6.00 a.m. The largest group was made up of contusions of the abdominal wall, none of whom died. Among the 56 patients with ruptured spleen, 31 had other injuries, such as fractured ribs and legs, and three had other organs injured. Peritoneal tap aided in the diagnosis in 16 cases and a leukocytosis of over 20,000 was noted in 30%. Delayed rupture occurred in 14%, and in one case, not until three months later.

There were 14 cases of injuries to the gastrointestinal tract: one perforation of the stomach, seven of the jejunum, three of the ileum, two of the colon and one of the duodenum.

The liver was injured in six and the cystic artery in one. There were four pancreatic injuries, all producing an elevated serum amylase level, and resulting in pseudocysts in three cases.

(Continued on page 345)

NEWS & VIEWS
ON THE ECONOMICS OF MEDICINE

Prepared by the Department of Medical Economics. The Canadian Medical Association

NUMBER 9

Our sources of information are private communications and published comments in medical journals and the lay press. These are usually reliable but incorrect quotation or interpretation is always possible.

The Department of National Revenue has recently released its analysis of taxation and incomes for the year 1958. The category of doctors and surgeons in private practice displaced engineers and architects as the highest professional earners.

12,201 doctors reported an average income of $15,264, an increase of $1,286 over 1957; average income of consulting engineers and architects was $14,260, lawyers were in third place at $13,163 and dentists fourth with an average income of $10,662. At the bottom end of the scale were nurses with an average income in 1958 of $2,342.(1)

While we do not consider that medicine need apologize for the level of income portrayed by these statistics, we should be aware that the tabulations are in some respects misleading and the comparisons incomplete.

The 12,201 doctors included represent only 65% of the approximately 19,000 graduate doctors in Canada in 1958. The remainder were interns, residents, salaried physicians or physicians who did not earn sufficient income to pay tax. Thus, the average income of all Canadian doctors would be considerably less than the level stated here.

Comparing income levels of professional groups in isolation from business and managerial categories is misleading. The incomes of senior salaried employees are not segregated for comparative purposes but are included in the very large category of salaried employees. Thus the salary of the company president is averaged with the salaries of his employees and the resultant composite average only indicates that there are far more employees than company presidents.

Similarly, doctors whose earned income is derived from salaried employment are classified not as "medical doctors and surgeons" but are buried in the large amorphous group of employees. This group of the profession represents close to 25% of the doctors of Canada and the proportion is steadily increasing. The returns of the voluntary survey of salaried doctors incomes conducted by the Section of Salaried Physicians just over a year ago indicate that their income is at a lower level than that of doctors remunerated by professional fees.

With these qualifications which come to mind when doctors are headlined as Canada's highest-earners, it is possible to conclude that

(over)

the medical profession is being reasonably rewarded for the essential, hardworking efforts which are expended in improving the health of our fellow citizens.

Premier Robichaud, the new New Brunswick premier, has announced that the premium method of paying for hospital insurance has been abolished. This announcement was expected in view of his pre-election platform pledge.

This step will necessitate other changes in the legislation passed by the previous administration. It is expected that these changes will be presented to a special session of the House of Assembly expected to convene some time in September.(2)

Premier Douglas of Saskatchewan made some interesting pronouncements at the recent Dominion-provincial conference in Ottawa. He stated— "The time has come to take a step toward a national prepaid medical care program." He urged that discussions start immediately on how to establish comprehensive health insurance at an early date.

He asked the federal government to take part in the plan for health insurance which he has proposed for his province and indicated that the federal government should provide federal assistance to any province which is prepared to introduce a prepaid medical program.(3)

British Columbia's Premier Bennett announced September 12th as the date for a general election in his province. The opposition C.C.F. party is expected to assiduously promote a prepaid medical program similar to Mr. Douglas' proposals in Saskatchewan.(4)

Premier Lesage of Quebec announced that his provincial health authorities met throughout the week of July 11th with federal representatives in efforts to establish a hospital insurance plan in Quebec by January 1, 1961.

He told a press conference that his government wants hospital insurance and not study committees. He announced that the committee set up by the Union Nationale administration a few months ago to study hospital insurance has been disbanded.(5)

REFERENCES:

(1) Toronto Telegram, July 27, 1960.
(2) Moncton Transcript, July 25, 1960.
(3) Toronto Daily Star, July 25, 1960.
(4) Toronto Globe and Mail, August 4, 1960.
(5) Toronto Daily Star, July 13, 1960.

(Continued from page 342)

Of the eight lacerated kidneys, five were treated by nephrectomy. There were 10 ruptured bladders, nine associated with fractures of the pelvis and five shorn off at the urethral junction.

Deaths were mostly among those with multiple injuries.

BURNS PLEWES

THERAPEUTICS

Permutation Trial of Diuretics: Chlorothiazide and Hydroflumethiazide.

F. KIIL: *Circulation*, 21: 717, 1960.

Chlorothiazide and hydroflumethazide in optimal doses (2 g. and 200 mg. daily) and placebo tablets were given, one drug on each of three days of study, in all permutations, to 12 patients treated by current methods for right heart failure.

Chlorothiazide showed a stronger diuretic action than hydroflumethiazide, whereas the natriuretic effects were not significantly different as judged by analysis of variance. Hydroflumethiazide was less kaliuretic.

The calculated sodium concentration in the additionally excreted urine during hydroflumethiazide administration was far above serum levels (the mean of 12 observations was 353 mEq./l.), and significantly higher than during chlorothiazide action. This effect was also demonstrated in six control patients during water loading, suggesting that hydroflumethiazide increases free water reabsorption when antidiuretic hormone is absent.

S. J. SHANE

DERMATOLOGY

On Sensitivity to Neomycin and Bacitracin.

V. PIRILA AND S. ROUHUNKOSKI: *Acta dermat.-venereol.*, 39: 470, 1959.

The authors report 184 cases of contact allergic dermatitis due to neomycin and/or bacitracin. This is the most common cause of contact dermatitis due to topical treatment in Finland. The use of neomycin as an intestinal sterilizer, a throat lozenge, and as packing in dental root infections caused a flare-up of the original neomycin dermatitis in one patient.

ROBERT JACKSON

PATHOLOGY

Analysis of the Clinical Application of the van den Bergh Reaction in Jaundice.

J. A. CHAPMAN, T. H. JOHNSON AND M. M. KARL: *Am. J. M. Sc.*, 239: 11, 1960.

The data and analysis in this paper support the conclusions of others that the determination of the direct serum bilirubin in conjunction with the total serum bilirubin does not materially aid in differentiating jaundice due to extrahepatic obstruction from that due to diffuse hepatocellular disease. Fractionation of serum bilirubin does, however, appear to be of value in differentiating hæmolytic anæmia from these. Patients with obstruction of the common bile duct have serum bilirubin elevation more marked than values noted in other liver disease, with obstruction due to carcinoma generally causing more marked elevation than that due to common duct stone.

In the opinion of the authors, routine use of quantitative fractionation of serum bilirubin does not seem justified.

S. J. SHANE

BOOK REVIEWS

THORACIC SURGERY BEFORE THE 20TH CENTURY. Lew A. Hochberg, Brooklyn, N.Y. 858 pp. Illust. Vantage Press, New York, Washington and Hollywood, 1960.

One of the interesting facets of medicine is its early history, and Dr. Hochberg has presented a masterful account of thoracic surgery before the twentieth century. The book contains many direct quotations and case reports which provide authenticity and interest to the reader. Primitive man recognized that the seat of life was in the thorax, and many famous surgeons throughout this period have written and contributed to thoracic surgical conditions.

Considerable research has been done in compiling this book and the presentation has been expertly arranged. A separate chapter on non-surgical contributions to the advancement of thoracic surgery has been included. A unique feature is the numerous illustrations which demonstrate the instruments and methods used in the various periods of medical history.

The excellent material and skilful presentation make this book a valuable contribution in the field of medical history.

THE MASSACHUSETTS GENERAL HOSPITAL, 1935-1955. Nathaniel W. Faxon. 490 pp. Illust. Harvard University Press, Cambridge, Mass.; S. J. Reginald Saunders & Company Limited, Toronto, 1959. $11.95.

This is a further instalment in the history of the Massachusetts General Hospital. It covers the period from 1935 to 1955 and it is in the main a series of annual reports arranged in chronological order. Thus it is easy to find the predominant hospital activity in any particular year. A comprehensive index further simplifies the problem of references.

Although this publication will appeal primarily to former interns and to grateful patients, it will also take the attention of those who have to direct the building and the management of a large modern hospital. For this hospital has an unexcelled record in the power to combine sound clinical medicine with basic and original research. Furthermore, it has been fortunate in being able to enlist the goodwill and the substantial financial support of a long list of intelligent and public-spirited men and women. The activities of these non-medical helpers form an impressive portion of the history of many medical advances.

The nature of this publication does not encourage sustained reading, but even casual browsing will bring to light some curious facts. Thus a sort of bed-pan jubilee was celebrated for an orderly who had given some 52 years of faithful service. One can find also the Massachusetts General Hospital version of the history of general anæsthesia. Perhaps the most curious fact is that in its early days the hospital allowed meat only on Wednesdays, Fridays and Sundays, which meant that those who wished to observe Lent had meat only once or twice weekly.

On the whole the book may be said to have a general as well as a parochial usefulness, but its market will be largely confined to those who have a sentimental regard for the hospital because it guided their stumbling feet when they started on the clinical road.

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NOUVELLE PRATIQUE CHIRURGICALE ILLUSTREE Fascicules XII, XIII et XIV. (New Illustrated Surgical Practice, Vols. XII, XIII and XIV.) Published under the direction of Prof. Jean Quénu by G. Doin & Cie, Editors, Paris, France, 1959. Each volume: $8.00 approx.

Cet ouvrage est connu depuis de nombreuses années par les chirurgiens de langue française comme manuel de technique chirurgicale publié à l'intention des étudiants en chirurgie et des chirurgiens; il est donc un classique de la littérature médicale française. La présentation actuelle ne diffère guère de l'ouvrage qui, auparavant était publié sous la direction de Victor Pauchet. Il s'agit, en somme, d'un atlas dans lequel chaque page est un dessin anatomique représentant un temps opératoire et accompagné de quelques notes descriptives. Les dessins sont semi-schématiques, très bien faits et très faciles à comprendre. De temps à autre un chapitre débute par les indications opératoires, les principes de la technique et la description des soins pré et post opératoires.

Dans le fascicule XII, nous avons retenu la technique de la lobectomie moyenne qui est expliquée avec beaucoup de détails. L'auteur recommande le drainage supérieur à travers une contre-incision scapulo-vertébrale, située au niveau de l'épine de l'omoplate. Ce point de technique n'est pas habituel dans nos milieux. Le chapitre sur l'hypertension portale est très bien fait et il y a des notes explicatives importantes sur la mesure de la pression portale, de même que sur la spléno-portographie peropératoire. L'anastomose porto-cave est décrite dans les détails de même que l'opération toute entière. Une autre technique intéressante décrite dans ce fascicule est celle de la pollicisation de l'annulaire. Cette méthode d'aborder le problème est ingénieuse et relativement nouvelle.

Dans le fascicule XIII, le chapitre sur la ligature des varices intra et extra-œsophagiennes est très bien fait. Ceci, plus la splénectomies avec anastomose spléno-rénale, que nous voyons un peu plus loin dans ce même fascicule, complète le traitement porto-systémique. On y trouve aussi une technique de lobectomie supérieure droite, pour cancer. Il s'agit, en somme, d'une lobectomie élargie.

Dans le fascicule XIV, l'auteur décrit sa technique de réparation d'une hernie hypogastrique. La technique décrite est une technique éprouvée. L'auteur recommande, cependant, l'emploi du crin de Florence, qui, dans nos milieux, a été complètement abandonné depuis assez longtemps. Par ailleurs, on recommande, avec raison, de drainer ces grands décollements, par un drain perforé attaché à une succion continue. Cette méthode est, sans aucun doute, préférable à l'emploi du drain cigarette ou d'une mèche, comme on le pratique habituellement. De plus, ce volume contient la description de plusieurs techniques de corrections orthopédiques qui nous paraissent excellentes.

En un mot, il s'agit là de manuels de techniques opératoires qui peuvent être très utiles, non seulement à l'étudiant en chirurgie, mais aussi aux chirurgiens qui désirent revoir rapidement une technique opératoire. Le seul reproche à faire porte sur la division de l'ouvrage. Les chapitres ne sont pas groupés par régions anatomiques ou par maladies, mais semblent avoir été laissés au hasard. Cette disposition rend la consultation de ces manuels assez difficile; même si la table des matières semble bien faite, il n'est pas toujours aisé de retrouver la technique désirée.

ATLAS DER GYNAKOLOGISCHEN OPERATIONEN (Atlas of Gynæcological Operations). O. Käser and F. A. Iklé. 451 pp. Illust. Georg Thieme Verlag, Stuttgart, W. Germany; Intercontinental Medical Book Corporation, New York, 1960. $35.25.

This book of 450 pages and 720 illustrations is not only another good atlas for gynæcological surgery but it also constitutes a very comprehensive source of information for the gynæcologist seeking advice in any operative situation.

The topography of gynæcological anatomy is tightly connected with the bladder, ureters and colon. Some gynæcological procedures, especially the operations for carcinoma of the cervix, may result in lesions of the urinary organs as a complication. For this reason particular emphasis is placed on the discussion of urological surgery.

The first chapter covers small diagnostic and therapeutic procedures and operations on the abdominal wall. The second chapter discusses the different abdominal operations on the uterus and adnexa, operations for the relief of pain, and operative procedures on the colon.

The third part includes vaginal operations on prolapsed genitalia, and vaginal operative approach on the uterus, adnexa, vagina and vulva. Radical and ultra-radical carcinoma operations follow. The closing chapter contains ample information on the urological and proctological operations including the urinary system, and treatment of exertional incontinence.

The introduction outlines preoperative and post-operative treatment of gynæcological procedures and action during the cardiac arrest.

Some of the articles deserve special comment. The discussion and comparison of the Gilliam-Doleris-Simpson operation with the Baldy-Webster-McCall method is excellent. Aldrige-Richardson's total hysterectomy, the technique of Cæsarean section, Manchester's operation for prolapsed uterus and radical operations of Wertheim and Schauta are described precisely and are vividly and amply illustrated.

Subdivisions of every chapter are furnished with a complete bibliography. It is the reviewer's opinion that this work of two famous Swiss gynæcologists will be a useful addition to the libraries of physicians reading German.

BASIC MEDICAL-SURGICAL NURSING. M. A. Mason. 513 pp. Illust. Brett-Macmillan Ltd., Galt, Ontario, 1959. $4.95.

The material in this book is clearly and simply presented and well illustrated. There is a good selection of references at the end of the chapters, as well as a section on suggestions for further study. A fairly detailed glossary is included at the end of the book.

From several standpoints there are considerable differences between the courses given to the practical nurses in the United States and those given to the nursing assistants in Ontario. This book undoubtedly was written for the former group. In some instances it could serve as a reference book but not as a textbook for the Canadian Nursing Assistants' Courses, as it is doubtful if this group of workers have sufficient background for the safe use of the material.

It is unfortunate that more material on nursing care and on adaptations for nursing care in the home was not included. The sections which give some detail on nursing care, for example, cerebral vascular accidents, should be very helpful.

A SHORT SYNOPSIS OF HUMAN PROTOZOOLOGY AND HELMINTHOLOGY. L. R. S. MacFarlane, London, 251 pp. Illust. E. & S. Livingstone Ltd., Edinburgh and London; The Macmillan Company of Canada Limited, Toronto, 1960. $3.60.

This small hard-covered volume is just as the title says, a short synopsis—and a very good one, too. Each organism is dealt with in a concise manner under well marked headings. One should not expect to find the formula for culture media for Entamœba or dosage schedules for drugs recommended for treatment. But the drugs most effective in the treatment of each parasitic infection are simply listed in order of preference. The illustrations are abundant and particularly good. This is an excellent little reference book for the clinical pathologist.

A HANDBOOK OF DISEASES OF THE SKIN. H. O. Mackey. 263 pp. Illust. 6th ed. Macmillan & Co., London, and St. Martin's Press, New York; C. J. Fallon Limited, Dublin, Ireland, 1959. $1.45 approx.

This well-known comprehensive small book has been partially revised and a number of recent advances have been included. An amazing amount of information is compressed in short chapters and a number of well-organized didactic summaries on anatomy, physiology and general symptomatology introduce the student to the basic foundations of the specialty. There are three short chapters on therapy, including a formulary, and a new chapter on dermatological nursing has been added.

The virtue of all-inclusiveness in short books tends to be marred by over-simplification and omissions. This book is not free of these faults. While esoteric entities are mentioned, and while, e.g., 24 lines are devoted to the rather ill-conceived entity of "acne frontalis", sclerodermas are described as skin changes only, without mentioning the more important localizations of this disease in heart, lungs and the intestinal tract. Likewise, various therapeutic procedures for this disorder are mentioned, including sympathectomy, ACTH and cortisone treatment, without any discussion of the special indications for and contraindications to these treatments.

It seems strange that the author often devotes equal space to rare and frequent disorders; the therapeutic suggestions reflect the personal experience of the author, mostly practical and logical but occasionally quite debatable and deviating from common practice. One is astonished to read that radium is recommended for the therapy of such a harmless condition as a seborrhœic wart.

There are a number of illustrations, most of them, but not all, satisfactory, and an excellent index.

While this book is full of useful information, it will be read to advantage by somebody with some experience in the field rather than by the undergraduate student.

CLINICAL OBSTETRICS AND GYNECOLOGY. A Quarterly Publication. Vol. 3, No. 1: Obstetric Emergencies, edited by M. L. Stone, and Pediatric Gynecology, edited by J. W. Huffman. 264 pp. Illust. Paul B. Hoeber, Inc., Medical Division of Harper & Brothers, New York, 1960. Subscription: $18.00 per year.

The March 1960 number of Clinical Obstetrics and Gynecology includes a symposium on pædiatric gynæcology. Since material in this area of pædiatrics is not too readily available, this new addition is very welcome indeed. It consists of contributions by experts in this field, and includes one on ovarian tumours in the premenarchal child by Dr. J. M. M. Darte of Toronto, who is the only Canadian contributor. There are chapters on the development of the female reproductive system during adolescence, symptomatic genital anomalies in childhood, vulvar disorders in premenarchal children, vulvovaginitis in childhood, vaginal and uterine tumours in childhood, the congenitally rudimentary gonad syndrome, dysmenorrhœa, dysfunctional uterine bleeding in adolescence, and delayed menarche. The articles are concise and well written, and represent the views of the authors, which are sometimes somewhat at variance with the views expressed in textbooks on endocrinology. In the opinion of one gynæcologist who discussed this book with the reviewer, the chapter on "The intersexed female", might have been improved if more details had been presented regarding the pathophysiology of the adrenogenital syndrome as well as a more detailed discussion on the various types of intersex. Objection was raised also to a consideration of presacral neurectomy in the discussion on dysmenorrhœa and menstrual disability. In the adolescent, the indication for presacral neurectomy must be extraordinarily rare if it ever exists at all. Since this book is to be read by general practitioners, as well as specialists, who see relatively few of these patients, it is possible that any suggestions that this therapy is useful in the adolescent may lead to many unnecessary operations. A chapter on gynæcological psychosomatic problems such as masturbation might have been worthwhile to provide the reader with a more complete symposium on pædiatric gynæcology.

BIOCHEMISTRY OF BLOOD IN HEALTH AND DISEASE. I. Newton Kugelmass. 543 pp. Illust. Charles C Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1959. $17.25.

This is really a textbook of biochemistry set up in such a way that the functions of the blood serve as a central theme. There are three main sections. The first two, "Blood as the transport system" and "Blood as the regulatory system", contain the subject matter which is found in most textbooks of biochemistry. "Blood as the defense system" is the third section and includes some hæmatology and immunology, as well as the chemistry of respiration and blood clotting.

It contains a tremendous amount of information. On almost every page there is reproduced at least one table, graph or diagram, and this is the book's most valuable asset. It is difficult for a reviewer to assess the worth and especially the accuracy of a text which covers such a wide field, just as it is difficult for one man to write critically about all the hundreds of topics found in biochemistry and clinical chemistry today. For example, the section on bilirubin is three years old and there have been important changes in that time. At the back of the book there is a very complete appendix of normal values which contains some internal inconsistencies such as this one: whole blood urea 20-40 mg. %, whole blood urea N 5-23 mg. % (urea = 2.14 urea N).

This volume represents a monumental effort but the reviewer believes that the day is past when a single author can produce an accurate text on so large and changing a field.

CURRENT THERAPY—1960. Latest Approved Methods of Treatment for the Practicing Physician. Edited by H. F. Conn. 808 pp. W. B. Saunders Company, Philadelphia and London, 1960. $12.00.

As stated by the author in the preface, this book endeavours to bring to the physician authoritative and factual information consistent with ready reference and practical application. It is a statement of methods currently employed by outstanding therapeutists. This book is a new edition and there are many new contributors. It should be of good reference value to all physicians.

FORTHCOMING MEETINGS

CANADA

PACIFIC DERMATOLOGICAL ASSOCIATION, Victoria, B.C., September 1-4. Dr. Edward J. Ringrose, Secretary-Treasurer, 2636 Telegraph Ave., Berkeley 4, Cal., U.S.A.

WORLD FEDERATION OF SOCIETIES OF ANÆSTHESIOLOGISTS, 2ND WORLD CONGRESS, Toronto, Ont., September 4-10. Dr. R. A. Gordon, Chairman of Organizing Committee, 178 St. George St., Toronto 5, Ont.

ONTARIO PUBLIC HEALTH ASSOCIATION, Toronto, Ont., October 3-5. Dr. G. K. Martin, Secretary-Treasurer, Room 405, 67 College St., Toronto, Ont.

CANADIAN SOCIETY FOR THE STUDY OF FERTILITY, Toronto, Ont., October 21 and 22. Dr. George H. Arronet, Secretary, Infertility Centre, Royal Victoria Hospital, Montreal, Que.

CANADIAN HEART ASSOCIATION AND NATIONAL HEART FOUNDATION OF CANADA, Toronto, Ont., November 30-December 3. Dr. John B. Armstrong, National Heart Foundation, 501 Yonge St., Toronto 5, Ont.

CANADIAN FEDERATION OF BIOLOGICAL SOCIETIES (Canadian Physiological Society, Pharmacological Society of Canada, Canadian Association of Anatomists, Canadian Biochemical Society), Fourth Annual Meeting, Ontario Agricultural College, Guelph, Ont., May 31, June 1 and 2, 1961. Dr. E. H. Bensley, Honorary Secretary, Canadian Federation of Biological Societies, Montreal General Hospital, 1650 Cedar Ave., Montreal 25, Que.

THIRD WORLD CONGRESS OF PSYCHIATRY, Montreal, Quebec, June 4-10, 1961. The General Secretary, III World Congress of Psychiatry, 1025 Pine Avenue West, Montreal 2, Quebec.

THE SOCIETY OF OBSTETRICIANS AND GYNÆCOLOGISTS OF CANADA—LA SOCIÉTÉ DES OBSTÉTRICIENS ET GYNÉCOLOGUES DU CANADA, Annual Meeting, The Chantecler, Ste-Adèle-en-Haut, Quebec, June 16-18, 1961. Dr. F. P. McInnis, Secretary, 688 Oriole Parkway, Toronto 12, Ont.

UNITED STATES

INTERNATIONAL CONGRESS OF PHYSICAL MEDICINE, Washington, D.C., August 21-26. Dr. W. J. Zeiter, 2020 East 93rd St., Cleveland, Ohio.

AMERICAN CONGRESS OF PHYSICAL MEDICINE AND REHABILITATION, Washington, D.C., August 21-26. Mrs. Dorothea C. Augustin, Executive Secretary, 30 N. Michigan Ave., Chicago 2, Ill.

NATIONAL CANCER CONFERENCE, AMERICAN CANCER SOCIETY, INC., AND THE NATIONAL CANCER INSTITUTE, Minneapolis, Minn., September 13-15. Dr. Roald M. Grant, Coordinator, 521 West 57th St., New York 19, N.Y.

INTER-SOCIETY CYTOLOGY COUNCIL, Chicago, Ill., September 23-25. Dr. Paul A. Younge, Secretary-Treasurer, 1101 Beacon St., Brookline 46, Mass.

AMERICAN SOCIETY OF ANESTHESIOLOGISTS, INC., New York, N.Y., October 2-7. Mr. John W. Andes, Executive Secretary, 188 West Randolph St., Chicago 1, Ill.

AMERICAN PUBLIC HEALTH ASSOCIATION, San Francisco, Cal., October 31-November 4. Dr. Berwyn F. Mattison, Executive Director, 1790 Broadway, New York 19, N.Y.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY, Chicago, Ill., October 9-14. Dr. William L. Benedict, Executive Secretary, 15 Second St. S.W., Rochester, Minn.

AMERICAN COLLEGE OF SURGEONS, Clinical Congress, San Francisco, Cal., October 10-14. Dr. William E. Adams, 40 East Erie St., Chicago 11, Ill.

ACADEMY OF PSYCHOSOMATIC MEDICINE, Philadelphia, Pa., October 13-15. Dr. Bertram B. Moss, 55 East Washington St., Chicago 2, Ill.

AMERICAN HEART ASSOCIATION, INC., St. Louis, Mo., October 21-25. Mr. Rome A. Betts, Executive Director, 44 East 23rd St., New York 10, N.Y.

AMERICAN COLLEGE OF GASTROENTEROLOGY, Philadelphia, Pa., October 23-26. Mr. Daniel Weiss, Executive Director, 33 West 60th St., New York 23, N.Y.

AMERICAN SOCIETY OF TROPICAL MEDICINE AND HYGIENE, Los Angeles, Calif., November 2-5. Dr. Rolla B. Hill, Executive Secretary, 3572 St. Gaudens Rd., Miami 33, Fla.

AMERICAN ACADEMY OF DERMATOLOGY AND SYPHILOLOGY, Chicago, Ill., December 3-8. Dr. Robert R. Kierland, Secretary-Treasurer, First National Bank Bldg., Rochester, Minn.

INTERNATIONAL ANESTHESIA RESEARCH SOCIETY, 35th Congress, Houston, Texas, April 9-13, 1961. A. William Friend, M.D., Executive Secretary, 227 Wade Park Manor, Cleveland, Ohio.

OTHER COUNTRIES

INTERNATIONAL CONGRESS OF INTERNAL MEDICINE (6th), Basle, Switzerland, August 24-27. The Secretariat, Sixth International Congress of Internal Medicine, Steinentorstrasse 13, Basle, Switzerland.

INTERNATIONAL CONGRESS ON DISEASES OF THE CHEST, sponsored by the Council on International Affairs, American College of Chest Physicians, Vienna, Austria, August 28-September 1. Mr. Murray Kornfeld, Executive Director, 112 East Chestnut St., Chicago 11, Ill.

INTERNATIONAL CONGRESS OF HÆMATOLOGY (8th), Tokyo, Japan, September 4-10. Organizing Committee, Science Council of Japan, Ueno Park, Taito-ku, Tokyo, Japan.

INTERNATIONAL CONGRESS OF PÆDIATRICS (10th), Lisbon, Portugal, September 9-15. Prof. Mario Cordeiro, Secretary-General, Clinica Pedietrica Universitaria-Hospital Santa Maria, Av. 28 de Maio, Lisbon, Portugal.

WORLD MEDICAL ASSOCIATION, 14th General Assembly and 63rd *Deutsche Arztetag*, West Berlin, Germany, September 15-22. Dr. Josef Stockhausen, Haedenkampstrasse 1, Cologne-Lindenthal, Germany.

INTERNATIONAL SYMPOSIUM OF CYBERNETIC MEDICINE (1st), Naples, Italy, October 2-4. Prof. Renato Vinciguerra, Secretary, Via Roma 348, Naples, Italy.

MEDICAL SOCIETY OF THE UNITED STATES AND MEXICO, Fifth Annual Meeting, Guadalajara, Jal., Mexico, November 8-10, followed by Mazatlan, Sin., Mexico, November 11 and 12. Dr. M. A. Carreras, 130 South Scott, Tucson, Arizona.

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GENERAL SURGEON, 35, married, Canadian graduate and internship. Completing residency in U.S.A. June, 1960. Eligible for Canadian certification and American Board of Surgery. Desires association with a surgeon or group. Willing to do some general practice. Reply to Box 795, CMA Journal, 150 St. George Street, Toronto 5, Ontario.

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CANADIAN F.R.C.S. in general surgery with thoracic training. Age 35. General practice and industrial experience. Association with established surgeon or group desired. Available September 1960. Reply to Box 906, CMA Journal, 150 St. George Street, Toronto 5, Ontario.

FEMALE PHYSICIAN.—Age 36, married, Europeon graduate, L.M.C.C., registered in Ontario, multilingual, wishes medical or medical administrative position with regular hours in Toronto. Reply to Box 931, CMA Journal, 150 St. George St., Toronto 5, Ontario.

OBSTETRICIAN/GYNÆCOLOGIST, F.R.C.S.(Eng.), M.R.C.-O.G., age 41, seeks opening where scope for energy, wide experience specialty and all round capability in general surgery. Married, family. Presently in United Kingdom. Reply to Box 941, CMA Journal, 150 St. George St., Toronto 5, Ont.

BRITISH WOMAN DOCTOR, age 50, wishes to contact Nova Scotia medical man in small town re partnership or sale of practice. Experience—25 years' general practice plus 15 years' gynæcology and obstetrics. In Halifax for registration on August 30th. Please reply to Dr. E. Bergmann, c/o Marco Bergmann Co., 599 11th Avenue, New York City.

Positions Vacant

ASSISTANT MEDICAL HEALTH OFFICER.—Applications are invited for the position of assistant medical health officer of the City of Regina. Applicant must be licensed to practise medicine in the Province of Saskatchewan or eligible for registration there. Should possess a diploma or master's degree in public health. Applications should state age, qualifications, training and date available. References should also be given. Applications and enquiries should be directed to the Personnel Department, City Hall, Regina, Saskatchewan.

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THE CANADIAN MEDICAL ASSOCIATION

JOURNAL

LE JOURNAL DE L'ASSOCIATION MÉDICALE CANADIENNE

Editorial Office—150 St. George St., Toronto 5

General Secretary's Office—150 St. George St., Toronto 5

SUBSCRIPTION RATES

The Journal is supplied to paid-up members of the Canadian Medical Association as a perquisite of membership. Medical libraries, hospitals, and individuals may subscribe to the Journal at $12.00 a year, payable in advance. There is a special rate for medical students residing in Canada of $2.50 a year. Subscriptions and all relative correspondence should be addressed to the Subscription Department, Canadian Medical Association Journal, 150 St. George Street, Toronto 5, Ontario.

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Manuscripts: Manuscripts of original articles, case reports, short communications, and special articles should be submitted to the Editor at the C.M.A.J. editorial office, 150 St. George St., Toronto, with a covering letter requesting consideration for publication in the *Journal*. Acceptance is subject to the understanding that they are submitted solely to this *Journal*, and will not be reprinted without the consent of both the Editor and the author. Articles should be typed on one side only of unruled paper, double-spaced and with wide margins. Carbon copies cannot be accepted. The author should always retain a carbon copy of material submitted. Every article should contain a summary of the contents.

The Editor reserves the right to make the usual editorial changes in manuscripts; these include such changes as are necessary to ensure correctness of grammar and spelling, clarification of obscurities or conformity to *Journal* style. In no case will major changes be made without prior consultation with the author. Authors will receive galley proofs of articles before publication, and are asked to confine alterations of such proofs to a minimum.

Reprints may be ordered on a form supplied with galley proofs.

References: Authors should limit references to published work to the minimum necessary for guidance to readers wishing to study the subject further. They should not quote articles they have never seen. Except in review articles, the maximum number of references should not be more than 25. References should be numbered in the text and should be set out in a numbered list at the end of the article, thus:

1. DOAKES, J.: *M. J. Kamchatka*, 1: 2, 1955, giving in order: (1) Author's name and initials in capitals. Where more than three authors are concerned in an article, only the first should be named, with *et al.* as reference to the others. (2) Quarterly Cumulative Index Medicus abbreviation of journal name. (3) Volume number. (4) Page number. (5) Year.

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PICKWICK, S., *Textbook of Medicine*, Jones and Jones, London, 1st ed., p. 30, 1955.

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MEDICAL NEWS *in brief*

(Continued from page 333)

THE RELATIONSHIP OF THE CANADIAN MEDICAL ASSOCIATION TO HOSPITAL PERSONNEL

As one of the prime objects of the Association is to improve medical services in Canada, it was not surprising that the Canadian Medical Association established standards of training for junior interns and laboratory technologists several years ago; and more recently for radiological technicians.

It has been traditional with members of the medical profession to give freely of their time in the training of other doctors and of ancillary medical personnel such as laboratory and radiological technicians. The free exchange of medical knowledge fulfils one of the tenets of the Hippocratic Oath taken by medical graduates.

C.M.A. Junior Intern Training Program

The intern training program is the oldest approval program of the Association, and was established to provide standards of training for new medical graduates. This was considered an important step in the training of the young doctors. The first year of training, following an intensive academic course, is the transition period—when the young doctor develops techniques and methods which will last him throughout his medical career. A well-organized and supervised junior intern training program is therefore considered essential, as the first experience in postgraduate medical education.

At the present time, there are 77 hospitals in Canada approved by the Association for junior intern training. These hospitals provide approximately 1200 internships for students who graduate from Canada's 12 medical schools each year.

Hospital appointments for the training of specialists are identified and approved by the Royal College of Physicians and Surgeons of Canada.

Laboratory Technologists

The role of the laboratory technologists in medical science is a most important one, and their skill, knowledge and training are in keeping with the desire of the Canadian Medical Association to improve standards of patient care in hospitals.

In 1941, the C.M.A., in co-operation with the Canadian Society of Laboratory Technologists, established a program for the approval of schools in general hospitals, for the training of laboratory technologists. This program was developed by a committee of the Association to ensure the supply of such properly trained personnel in our hospitals and public health laboratories.

At the present time, there are approximately 100 approved hospital laboratory schools in Canada; and the standard of training is approved by the Canadian Medical Association Committee on Approval of Schools for Laboratory Technologists.

Approved schools of training are located in all provinces; and students registering in a hospital training program are required to

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MEDICAL NEWS *in brief*
(Continued from page 26)

have their senior matriculation, including chemistry, either physics or a biological science, and two mathematics. Students may be trained in a general course or take special courses in such subjects as histology, serology, bacteriology, and hæmatology. Graduates from schools approved by the C.M.A. are eligible to write the Canadian Society of Laboratory Technologists' examination, which leads to the qualification of REGISTERED LABORATORY TECHNOLOGIST, R.T.(Can.).

In addition to the 12-24 months' hospital training program, several Canadian universities provide certificate and degree courses. In order to qualify for registration, however, students must take practical training in an approved hospital laboratory.

The total number of registered technologists presently employed in Canadian hospitals and other institutions is over the 1500 mark. Following completion of the first year in the training program, 36 qualified by registration; and in 1959, 375 students graduated.

Although the number of graduating students has more than doubled since 1947, there is still an acute shortage of skilled personnel to meet the demand.

Radiological Technicians

In 1959, the Canadian Medical Association accepted the responsibility for approving schools for training radiological technicians. For some years, the approval of these schools and provision of training standards were carried out by the Canadian Society of Radiological Technicians and the Canadian Association of Radiologists. The C.M.A. entered the field at the request of these organizations.

In 1958, the Joint Committee on Technical Training for the C.S.R.T. and the C.A.R. became known as the "Joint Council on Technical Training School Standards"; and six members of this Council are now represented on the C.M.A. Committee on Approval of Training Schools for Radiological Technicians.

The purpose of this approval program is similar to the others, and is to improve and maintain standards of education and performance of radiological technicians, and to ensure their supply in Canadian hospitals, offices and clinics. Training is supervised by the radiologist in charge, as well as properly trained registered technicians.

Students applying for admittance to the approved training schools are required to have at least their junior matriculation, which includes physics and chemistry; biology is considered helpful but not essential. The length of the course is two years. Similar to laboratory technologists, graduates of approved schools are eligible to write examinations of the C.S.R.T. and, if successful, to obtain their R.T. qualifications as a properly trained radiological technician.

At the present time, there are over 100 interim approved schools of training. In the main, they are affiliated with general hospitals, cancer clinics and offices of radiologists. Last year some 291 students successfully passed the qualifying examinations. In 1959, there were only 79 graduates, but

during the past ten years there have been over 1800.

Although the supply of qualified radiological technicians has been reported as being adequate in some areas, there are definite indications that the number of male graduates is not keeping pace with the demand. An underlying cause seems to be a relatively low rate of remuneration, which does not compete with other vocations.

The Canadian Medical Association recognizes the need to increase the tempo of the approval programs—in order to meet the increasing demand for these services under the current provincial hospital programs.

THE CASE AGAINST BUERGER'S DISEASE

A careful study was made of the clinical records of all patients with peripheral arterial occlusive disease admitted to the Beth Israel Hospital, Boston, from 1928 to 1956, inclusive. Wessler *et al.* (*New England J. Med.*, 262: 1149, 1960) review the history of Buerger's disease, the pathological examination of all amputated limbs, the superficial vein biopsies and the autopsy material in patients with the onset of arterial insufficiency before the age of 45. After exhaustive study, the conclusion was reached that the disease which was originally described by Buerger is indistinguishable from atherosclerosis, systemic embolization, or peripheral thrombosis singly or in combination.

It appears that thromboangiitis obliterans (Buerger's disease) cannot be considered an entity in either a clinical or a pathological sense and the authors recommend that the term be discarded.

WHO COMMITTEE STRESSES NEED FOR BOTH SALK AND LIVE POLIO VACCINES

The WHO Expert Committee on Poliomyelitis met in Washington last June under the chairmanship of Professor Stuart Harris of the University of Sheffield, England, to consider progress in the control of polio by vaccination. The meeting followed a week's conference on live polio vaccine convened jointly by PAHO and WHO.

In its report it emphasized that both the inactivated Salk vaccine and the live vaccine prepared from attenuated viruses had a major role to play in the control of polio. The Committee pointed out that the Salk vaccine had achieved over 90% success in conquering paralytic polio when it had been properly used, but that it was expensive and difficult to administer, especially in countries where health services were not well developed. Especially in these areas the live vaccine given by mouth as a liquid or in candies would be easier and more economical to use.

The Committee considered that the live vaccine had now been proved safe for the immunization of children in extensive trials in America, Africa and Eastern Europe. More than 60 million had been vaccinated in the U.S.S.R. alone without reported ill effects. Because of lack of data, the Com-

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new

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MEDICAL NEWS *in brief*

(Continued from page 31)

mittee reserved judgment about its widespread use in countries where polio affected a higher proportion of adults. This was because polio infection is more risky in adults who have never been in contact with the virus and it was thought that the live vaccine had not yet been adequately tested for safety in this highly susceptible group. The Committee advocated Salk vaccination for primary immunization in parts of North America, and Northern Europe and in countries where adults often contract the illness, but thought that the live vaccine could be used as a booster. The Committee pointed out that the virus in the vaccine, although safe when given to children, spreads and infects close contacts; normally it immunizes them without harm, but it is known that it sometimes changes a little in the process and might therefore become more likely to cause symptoms in specially susceptible persons. There is little evidence that this has occurred in the course of giving the vaccine to many tens of millions, but the Committee felt that this point had not been fully established.

The Committee also pointed out that information on the effectiveness of the vaccine, although hopeful, was not conclusive as to its degree under all circumstances. So far this had certainly not proved to be 100%. A complicating factor in the effectiveness of these vaccines is the blocking effect of interfering intestinal infections caused by viruses circulating in the community at the time of administration, a circumstance frequently encountered in tropical areas. However, progress towards the solution of this problem is being made. In this regard, the Committee stressed that the new vaccine could not be relied on to give full protection in one dose and that repeated administrations are needed.

Although hopeful for the future, the Committee stressed that many problems remained to be solved regarding the preparation, testing and use of these vaccines. One problem which was highlighted was the elimination of other viruses which may be difficult to detect in the cultures of monkey-kidney tissue in which the vaccine virus is grown.

OBSERVATIONS ON PRIMARY ALDOSTERONISM

The clinical features in two patients with primary aldosteronism reported by Cortes, Shuman and Channick (*Am. J. M. Sc.*, 239: 324, 1960) were entirely dissimilar and did not coincide with the syndrome described in many of the published cases. The first patient, a 45-year-old Negress, under treatment for hypertension for five years at another hospital, was admitted in a state of peripheral circulatory collapse after an acute episode of diarrhœa. Hypokalæmia, attributed initially to diarrhœa, proved resistant to administration of potassium over a period of several weeks. She had no symptoms of weakness, paralysis, or tetany before admission.

The second patient, a 50-year-old white woman, was admitted

for treatment of menorrhagia. Hypertension and œdema had been under treatment for four years. On two occasions, administration of diuretics had resulted in brief episodes of extreme muscular weakness.

In both cases, retroperitoneal CO_2 insufflation demonstrated an enlargement of the left adrenal gland. Twenty-four-hour urine specimens revealed elevated aldosterone levels (80 μg. and 24 μg. respectively) with normal 17-ketosteroid and 11-oxysteroid excretion. Surgical removal of the adrenals in both cases revealed almost identical adenomas. Large amounts of potassium salts were required to maintain normal serum levels of this electrolyte during the operative period. Postoperatively, the blood pressure declined to shock levels in one case and to normal in the other; in both, there was a transient period of hypertension during cortisone administration after operation. Abnormal glucose tolerance tests reverted to normal after recovery.

PERIODIC MEDICAL EXAMINATIONS AFTER OPERATION FOR CARCINOMA OF THE COLON OR RECTUM

Two cases are reported by Scudamore, Corr and Judd (*Proc. Staff Meet. Mayo Clin.*, 35: 258, 1960) to illustrate the value of periodic medical examinations after operation for carcinoma of the colon or rectum.

A survey was conducted of 507 patients in whom a diagnosis of carcinoma of the colon or rectum was made during a single year, to evaluate the worth of periodic examinations. Only 333 of these patients had had operations that might definitely be considered "curative", and 213 of these returned for regular follow-up examinations. The five-year survival rate for this select group was 59.9%. Recurrent lesions amenable only to palliative therapy, or none at all, were discovered in 57 patients on interval examination; adenomatous polyps that could be fulgurated or excised were diagnosed in 24 patients; and new, recurrent or metastatic neoplasms that could be treated surgically were found in 19 patients.

The results of this study justify the recommendation to patients who have undergone operation for carcinoma of the colon or rectum that they have a medical examination at least once a year for the rest of their lives.

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SOME PRACTICAL ASPECTS OF THE SMOKING-CANCER PROBLEM

The majority opinion favours smoking as a cause of cancer of the lung. Wynder and Hoffmann (*New England J. Med.*, 262: 540, 1960) investigated means of avoiding or reducing the risk to the smoker. Laboratory investigation was carried out on leading American brands of cigarettes to determine the amount of smoke condensate they produced when smoked with an artificial smoking machine. The condensate was analyzed for its content of benzo-α-pyrene and nicotine. Studies were carried out on human subjects to determine the amount of fluorescent substances absorbed by the lung after inhalation of smoke. Of a group of 304 cigarette smokers, all men

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over 40 years of age, 43% had changed to filter cigarettes. There was a marked improvement in the statistical incidence of symptoms from smoking, such as cough, with the use of filter cigarettes. Laboratory investigation had shown that filter cigarettes produced much less smoke condensate than the ordinary unfiltered type.

The authors suggest the following measures to reduce the risk of cancer of the lung in smokers: moderation for those who can not give up smoking, use of filter cigarettes with the lowest yield of smoke condensate, avoidance of smoking to the butt since there is more smoke condensate in the latter part of a cigarette, and avoidance of deep inhaling of cigarette smoke.

VARIATIONS IN SERUM LIPID CONCENTRATION AND CLINICAL CORONARY DISEASE

Variations in serum cholesterol level seem to be more closely related to clinically recognized coronary artery disease than is the five-year average. In 16 cases of myocardial infarction, Groover, Jernigan and Martin (*Am. J. M. Sc.*, 239: 133, 1960) found that a period of abnormal fluctuation preceded each attack. These variations were usually above the five-year average except in three cases in which the fluctuations were diphasic for a period of several weeks before the characteristic upward variations became pronounced.

Cholesterol, in the opinion of these workers, seems to be as valuable as any of the other lipid elements for determining the variations in concentration, except for the fact that the variations in total lipoproteins occur earlier and are of greater magnitude than those for cholesterol. Any one determination is valueless in determining the changes in one individual developing clinical manifestations of coronary disease. A sufficient number of laboratory values must be available to indicate a trend.

CANADA SHIPS MILLIONTH CURIE OF RADIOACTIVE ISOTOPES

With the export of a Theratron B Cobalt^{60} Unit and Source to Brazil last June, the Commercial Products Division of Atomic Energy of Canada Limited has shipped the millionth curie of Canadian-produced isotopes. These shipments have aided medicine, industry and research throughout the world including countries in the Soviet block. No fewer than 50 countries have obtained isotopes produced in the NRX and NRU reactors at Chalk River.

Cobalt^{60} for cancer treatment accounts for over 500,000 curies of this 1,000,000 curie total. It is estimated that this cobalt provides more than 100,000 cancer treatments each month. Other uses of

isotopes, largely industrial, include such widely diversified applications as studies of the effects of radiation on plastics, textiles and other materials, radiographic inspection of metals, insect control and the sterilization of pharmaceutical and medical supplies.

Since the first shipment, 12 years ago, C.P.D. has made over four thousand shipments of radioactive material.

BRONCHOGENIC CARCINOMA: A COMPARISON STUDY

Of 547 consecutive proved cases of bronchogenic carcinoma with complete follow-up analyzed by Hughes, Pate and Campbell (*J. Thorac. & Cardiovasc. Surg.*, 39: 409, 1960), the median age of the patients was 58 years. The average duration of symptoms before hospitalization was over eight months, and 16% of the patients had symptoms for over two years before being admitted. Seven per cent of the patients had abnormalities by chest x-ray studies in the absence of symptoms; 65% had a mass demonstrated in roentgenograms of the chest, and these masses exceeded 6 cm. in diameter in 73% of cases. The operability rate was 41.4% of all patients admitted; the resectability rate was 60.3% of those patients operated upon. Lobectomy in properly selected cases gave results comparable to those of pneumonectomy. The operating room mortality was less than 1% and the 30-day mortality slightly over 2%. The five-year survival rate after resection was 37%. High-voltage x-ray and nitrogen mustard therapy were found to have a place in prolonging useful life and in palliation. The five-year survival rate of all patients with bronchogenic carcinoma originally admitted to the hospital concerned was 11.5%.

RESECTIONAL THERAPY FOR PULMONARY TUBERCULOSIS

Kimel analyzed 125 cases of combined segmental and wedge resection with reconstitution, and a similar group of 223 cases without reconstitution in regard to postoperative complications as determined by the incidence of space problems, fistulas, empyema, and occult fistulas (*J. Thorac. & Cardiovasc. Surg.*, 39: 405, 1960). A smaller group of pure segmental resections was similarly studied. He found that there was a significantly higher incidence of major complications and space problems in segmental resections without reconstitution. A large percentage of the complications were encountered in the presence of good antimicrobial coverage and were due to infection by secondary organisms. The conclusion is drawn that the anatomical situation, comprising the conical segmental defect with raw lung surface exposed to pleural cavity, plus secondary contamination from air leaks, is favourable to the development of secondary infections and should therefore be avoided. It appears that reconstitution of raw lung surfaces presents a method of avoiding the undesirable anatomical situation, of reducing serious air leaks and, therefore, of reducing markedly the incidence of serious postoperative complications.